THE PLANT DISEASE REPORTER

Issued By

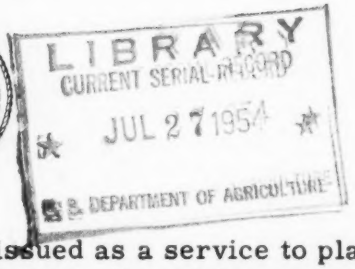
THE PLANT DISEASE SURVEY

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 38 Number 6

June 15, 1954



The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Section of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

Issued by

SECTION OF MYCOLOGY AND DISEASE SURVEY

Horticultural Crops Research Branch — Plant Industry Station, Beltsville, Maryland
Volume 38 — June 15, 1954 — Number 6

IN THIS ISSUE

Katharina Bollenbacher and Paul B. Marsh describe a fluorescent-fiber condition in raw cotton, associated with various organisms, especially Aspergillus and Rhizopus, page 375.

Selective cutting when henequen leaves are harvested results in a progressive reduction in amount of black leaf spot, an important disease in El Salvador, according to Bowen S. Crandall et al., page 380.

N. J. Whitney reports a consistent relationship between bird damage and ear rots in hybrid corn in Ontario, page 384.

G. H. Boewe sends the sixth annual prediction of occurrence of bacterial wilt of corn in Illinois, page 388.

E. D. Hansing summarizes 1953 tests of new compared with older fungicides for the control of cereal smuts in Kansas, page 389.

L. A. Brinkerhoff et al. discuss the effects of chemical seed treatments on nodulation of legumes, page 393; they used soybeans, peanuts, and cowpeas in their tests.

Harold J. Jensen and Glen E. Page describe an experimental blade-type soil fumigant applicator, page 401.

Several species of Chaetomium were associated with a foliage disease of greenhouse-grown potatoes in South Dakota, according to Allyn A. Cook, page 403.

Robert A. Conover reports good control of bacterial spot on tomato and pepper seedlings in Florida with Agrimycin, page 405.

H. Rex Thomas and H. H. Fisher describe a rapid method of testing snap beans for resistance to the Common Bean Mosaic virus, page 410.

H. H. Thornberry, reporting studies on insect transmission of the Eastern Peach X-Disease virus in Illinois, page 412, suggests an explanation for the lack of natural spread from peach to peach or chokecherry.

Observations reported from Missouri by Robert N. Goodman, page 414, show that apple fruits can serve as a source of overwintering fireblight inoculum.

The rose black mold fungus caused severe losses of evergreen grafting stock in a New Jersey nursery, according to Charles E. Hess and D. S. Welch, page 415.

Roy A. Young emphasizes the ease with which plant pathogens may be disseminated with nursery stock, and the serious consequences that follow, page 417.

Unusual records of plant diseases, page 421: Outbreak of Pythium rot in newly set flue-cured tobacco in Virginia, by Wilbert A. Jenkins; Tobacco plant situation in Southwest Virginia, by S. B. Fenne; Potato wart intercepted from Bolivia, by Paul X. Peltier; Destructive outbreak of loose smut in a Georgia wheat field, by T. D. Persons.

Publication of the Proceedings of the Seventh International Botanical Congress, Stockholm, Sweden, 1950, page 422.

April weather, page 423.

CONTRIBUTORS PLEASE NOTE; For suggestions as to content and preparation of articles, illustrations, etc. for the Reporter, see PDR 35 (2): 121-122, Feb. 15, 1951; 36 (5): 214, May 15, 1952; and 36 (12): 494, Dec. 15, 1952.

Vol. 38, No. 6--PLANT DISEASE REPORTER--June 15, 1954

A PRELIMINARY NOTE ON A FLUORESCENT-FIBER CONDITION IN RAW COTTON

Katharina Bollenbacher and Paul B. Marsh[1]

Abstract

Several samples of raw cotton containing yellowish spots which fluoresce strongly in ultra-violet light have been examined in respect to the microbial population present. Aspergillus flavus was found with very considerable regularity on the fluorescent fiber. Aspergillus niger and Rhizopus sp. were also commonly present, while various other organisms were found somewhat less frequently.

The existence of damage in the fluorescent fibers was indicated by their high alkali-swollen fiber width and by the presence of helical splitting.

A bright fluorescence similar in appearance to that of field samples has been produced by incubating cotton locks taken from unopened bolls with A. flavus. Not all locks have fluoresced, however, even after supporting heavy fungus growth. Some as yet unknown internal factor in the lock appears to be pre-requisite to the production of the fluorescent condition. Helical splitting and high alkali-swollen fiber width also occurred in such incubated samples. Further work is in progress on several aspects of the fluorescent-fiber problem.

Introduction

In late 1953 and early 1954, several reports were received at Beltsville concerning a yellow spotting of raw cotton in the 1953 crop and it was thought desirable to investigate the phenomenon. In some cases it had been noted that the fiber exhibited a strong fluorescence under ultra-violet illumination. Although the phenomenon is not as yet entirely understood, a number of relevant observations have been made which may be of use or interest to others concerned with the problem. Some of these observations are described in the following initial progress report.

In November, 1953, a sample of seed cotton and open bolls from the 1953 crop at Blythe, California, was sent to Beltsville by Mr. Stuart Evans of Arizona Fertilizers, Inc., Phoenix, Arizona, with a request for diagnosis of the cause of a yellowish stain on the fiber. The stain was said to have developed in rank cotton which was approximately six feet tall, and to have been confined to the bolls on the lower 2 to 3 feet of the plant. It occurred on several varieties and was believed to have developed during August, at a time when most of the lower bolls were mature and had just started to open. The problem was regarded as serious. As an illustration of the difficulty being experienced, it was stated that on one 1000-acre ranch, 50 percent of the bales harvested had been classified "spotted" as a result of the yellow staining. A condition similar to that at Blythe was said to have been observed also in the Yuma, Arizona area.

Micro-organisms Present on the Affected Fiber

Upon examination, the Blythe sample was found to exhibit yellowish spots which fluoresced with a brilliant greenish yellow color under ultra-violet light ("Mineralight" Model V-41). There was much mycelial growth on the fluorescent fiber and helical splitting of the fiber was observed (5). Many of the locks were incompletely fluffed and the fiber could be pulled off the seed very easily. In many instances the basal portion of the lock was compact, while the remainder of the lock was more or less fluffed. The yellow color and the fluoresence were not localized on the surface of the lock but, on the other hand, frequently occurred to the base of the lint fibers and also on the fuzz fibers of the seed as well. The carpels were free from the gross distortion and sticking of the fiber to the inner carpel wall which is characteristic of attack by cotton stainer insects (1). Insect transmission might, however, have been involved in early or primary cycles of infection without being apparent from the sample as sent to us. The fluorescent fiber was obviously much weakened, so much so as to render impossible its satisfactory preparation into a bundle for a strength test.

Observations on the sample as received disclosed the presence of dried Aspergillus spore heads on the fiber, on the outer surfaces of the carpels, and on the pedicels. When these

[1] Field Crops Research Branch, Agricultural Research Service, United States Department of Agriculture.

spore heads were transferred to a nutrient agar medium, typical Aspergillus flavus fruiting structures were produced. When tufts of the yellow fiber were incubated on water-agar, the predominant organisms which grew were Aspergillus flavus[2], a Rhizopus, and a yellow bacterium. Seeds from fluorescent locks were surface-sterilized with sodium hypochlorite solution, washed in sterile water, and cut apart with a sterile scalpel. The resulting seed fragments were then placed on water-agar and incubated for several days, at which time a very heavy growth of Aspergillus flavus appeared on a majority of them. Other fungi present on a few of the fragments were Fusarium sp. and a Mucor.

In the course of a visit at Beltsville in December, 1953, Mr. Edwin Jennings of Anderson, Clayton and Co., Houston, Texas, indicated that he had seen fluorescent spots in samples from baled cotton. Subsequently, eight fiber samples were kindly forwarded by him to Beltsville at our request. It was noted that the rather numerous fluorescent spots in these samples were similar in appearance to those in the Blythe sample. From these samples, 25 small tufts of fiber were selected from 25 fluorescent spots and the fluorescent tufts incubated on water-agar. Aspergillus flavus was prominent in the growth originating from more than half of these tufts and was isolated from 24 of them. In cultures of 14 of the isolates very hard blackish-brown sclerotia similar to those described for A. flavus (11) developed. The bales from which these samples were taken were known to have come from California, the fiber very probably originating from plants grown in 1953.

Fluorescent fiber from several samples from other sources was incubated similarly on water-agar in Petri dishes and examined in the usual way, placing the dishes on the stage of a compound microscope. A summary of the micro-organisms observed on the various samples is presented in Table 1. All of the samples listed in the table are belived to have originated from the 1953 crop, with the exception of the Rainwater sample, which was grown in 1952, and the Nickerson sample, which was over 10 years old. The Aspergillus flavus seemed noteworthy in the sense that among the several fungi found it was the only one which had not been noted frequently on raw cotton before.

Alkali-Swelling of the Affected Fiber

Damage to a cotton fiber is frequently evidenced by an abnormally high degree of swelling in alkali (6). Fluorescent fiber from several samples was swollen in 18% NaOH at 20° C. and the alkali-swollen fiber width measured by the use of a micrometer eyepiece. In each case, non-fluorescent fiber from the same sample was similarly observed for purposes of comparison. The results, shown in Table 2, indicated clearly that the fluorescent fiber had been damaged.

Production of Yellow Color and Fluoresence in Cotton by Incubation with Aspergillus flavus

Since the yellow staining at Blythe was stated to have occurred in very tall, rank cotton and particularly on the lower bolls, it seemed reasonable to believe that the fiber had never dried out at the time of boll opening before becoming infected. Consequently, undried locks were used in laboratory incubations. The locks were taken with aseptic precautions from unopened cotton bolls, placed without prior drying into sterile Petri dishes containing mats of wet absorbent cotton to maintain a high humidity, inoculated by a small sterile brush with a pure microbial culture, incubated for ten days and then examined for fluoresence under the ultraviolet lamp.

In the above experiments it was noted that an Aspergillus flavus isolate from the Blythe sample was capable of causing the fiber in the incubated locks to become yellow and fluorescent. The fluorescence produced was a bright greenish yellow, similar in appearance to that originally noted in the Blythe sample. Some of the incubated locks, however, did not develop fluorescence, even though supporting a heavy fungal growth. The bolls used for the experiments came from greenhouse-grown plants and were of varying maturity; failure to obtain fluorescence in some cases seemed to be associated with immaturity of the boll. Control locks incubated without inoculation consistently failed to develop fluorescence. The purity of the cultures used for the inoculation was checked and confirmed by the streaked plate method. The

---

[2] Thanks are due to Miss D. I. Fennell, USDA Northern Utilization Branch, Peoria, Illinois, for confirming the identification of the fungus, an isolate of which has been entered into the Peoria collection as #A-5501.

Table 1. Micro-organisms observed on fluorescent cotton fiber after three days incubation of the fiber on water agar.

Source of sample and designation	Location of growth	Micro-organisms observed on fiber: Actinomyces	Alternaria sp.	Aspergillus flavus	Aspergillus niger	Cladosporium sp.	Fusarium sp.	Rhizopus sp.	Other organisms
Mr. Stuart Evans[a]	Blythe, Calif.	0	1	5[h]	1	0	1	3	Bacteria 2
Mr. Ed. Jennings[b]	California								
39429	"	0	3	3	2	3	0	0	
41746	"	1	2	4	3	2	2	2	Verticillium sp. 1,
41750	"	2	1	3	3	4	1	0	
41761	"	0	0	1	1	0	0	2	
43024	"	2	1	3	2	1	1	0	
43029	"	2	2	2	1	0	1	1	Verticillium sp. 1, Asp. terreus 2.
DVOS		0	4	1	4	0	0	0	
"Calif. cotton"		2	0	4	2	0	0	1	
Mr. K. T. Louis[c]									
1	Arizona	1	0	3	4	0	0	2	
2	Ashdown, Arkansas	0	0	0	0	0	0	1	Bacteria 4.
3	Unknown	1	2	4	2	0	0	2	
4	Vernon, Texas	2	0	3	2	0	3	0	Bacteria 3, Asp. terreus 2.
5	Sulfur Spring, Texas	0	0	1	1	0	2	0	Verticillium sp. 2, Bacteria 2.
Dr. J. P. Elting[d]									
1	Arizona	3	0	0	0	0	0	0	0
2	"	0	0	2	2	0	0	1	0
3	"	0	1	4	3	0	2	3	0
4	"	0	2	4	3	0	3	2	0
Mr. C. F. Rainwater[e]	San Benito, Texas	2	0	3	2	0	0	3	0
Miss D. Nickerson[f]									
1	Unknown	1	0	1	0	0	0	0	0
2	"	0	0	1	0	0	0	0	0
3	Brownsville, Texas	0	0	0	2	0	0	0	0
Mr. O. Niles[g]	California	1	1	4	3	0	1	1	Bacteria 1, Verticillium sp. 1.

[a] Arizona Fertilizers, Inc., Phoenix, Ariz.
[b] Anderson, Clayton and Co., Houston, Texas.
[c] Otto Goedecke, Inc., Hallettsville, Tex.
[d] Kendall Mills, Charlotte, N. C.
[e] U. S. Dept. of Agriculture, Beltsville, Md.
[f] U. S. Dept. of Agriculture, Washington, D. C.
[g] Calcot Ltd., Bakersfield, Calif.

[h] The numbers represent an estimate of the amount of each organism present, in the sense 0=none, 1=trace, 2=light, 3=medium, 4=heavy, 5=very abundant.

Table 2. Alkali-swollen width of fluorescent fibers in comparison with non-fluorescent fibers from the same samples.

Source of sample and designation	Width in microns[a]		
	Fluorescent fibers	Non-fluorescent fibers	L.S.D.
Mr. Stuart Evans			
Blythe sample	35.6	25.4	0.8
Mr. Edwin Jennings			
41746	31.4	25.3	0.4
43029	30.7	25.0	0.4
"Calif. cotton"	29.0	24.9	0.3
Mr. C. F. Rainwater	30.7	25.3	0.3
Dr. J. P. Elting			
Sample 3	32.3	24.7	0.5
Sample 4	32.7	24.8	0.4
Mr. K. T. Louis			
Sample 1	32.0	24.7	1.0

[a] Each figure is an average from measurement on 100 fibers.

fiber in the A. flavus-incubated locks exhibited pronounced strength loss, helical splitting, and an alkali-swollen fiber width of approximately 29 microns, as compared with 24 microns for fiber from a sterile-incubated lock.

When a Rhizopus isolated from the Blythe sample was incubated on cotton locks in a manner similar to that employed with A. flavus, the aerial mycelium of the fungus exhibited a greenish-blue fluorescence but the fiber was not fluorescent; the fluorescent color was distinctly different from that of the field samples. A yellow bacterium from the Blythe sample grew on the fiber but there was no fluorescence.

Aspergillus flavus NRRL #482 (described in Thom and Raper's "Manual of the Aspergilli", pp 263-266) was brush-inoculated upon undried locks. After ten days incubation, locks from five different bolls showed the greenish-yellow fluorescence, while locks from seven bolls had growth but no fluorescence. The aqueous-extract pH of the incubated fiber was in the range of 6.2 to 6.6, with no correlation between the pH and fluorescence. Fluorescent fiber from the Blythe sample also exhibited a pH in the same range. On the other hand, ordinary raw cotton lint incubated in a saturated atmosphere for periods up to 12 weeks with A. flavus developed a pH in the range of 9.5 to 10.0 and showed no fluorescence.

Discussion

Yellow spots have been observed in cotton over a period of years by cotton classers and others. The percentage of the total crop affected is said to have been variable from year to year and never very large, most of the affected fiber being western in origin. At Kendall Mills, the striking fluorescence of certain spots in ultra-violet light was observed some years ago, the phenomenon being regarded as a curiosity of unknown origin (2). Jennings (3) and others, have also seen the brightly-fluorescent spots. Our observations to date neither support nor refute Jennings' (3) suggestion that the fluorescent spots are related in some causal fashion to the presence of aphid honey-dew on the fiber, although it is clear that they may be produced experimentally with neither aphids nor honey-dew present. Others may be expected to observe fluorescent spots in cotton in the next few months as a result of the fact that detection of heat damage by examination of the fiber under ultra-violet illumination is of current interest in several laboratories. It should be noted that in addition to the brightly fluorescent spots here discussed there are also spots which exhibit a pale whitish fluorescence. The origin of these latter spots is at present unknown to us.

Aspergillus niger and Rhizopus nigricans are well known as causes of boll rots of cotton in California and Arizona (9) so that their occurrence in the samples under study in the present work is not surprising. Aspergillus flavus, on the other hand, is relatively or possibly completely unrecognized in the literature in respect to its growth on cotton fiber in the boll. An interesting feature of the literature relating to this fungus on cotton fiber is the fact that it has been regarded as a "non-cellulolytic" micro-organism in the sense of having failed to bring about strength loss of cotton fabric incubated with it (4, 7, 8, 12). It should be mentioned in this connection that the cellulose in undried cotton fiber is in an amorphous condition, and that amorphous cellulose is generally regarded as more susceptible to microbial and enzymatic action than crystalline cellulose (10). No inconsistency necessarily exists therefore between the results here reported and previous data on the action of A. flavus on cotton fiber.

ACKNOWLEDGEMENT: Thanks are expressed to the several individuals mentioned in the text and tables for their cooperation in supplying samples and to Dr. J. T. Presley for his aid in the procurement of facilities for carrying out the work.

Literature Cited

1. Cassidy, T. P. and T. C. Barber. 1939. Hemipterous insects of cotton in Arizona: their economic importance and control. Jour. Ec. Entomology 32: 99-104.
2. Elting, J. P. 1954. Personal communication.
3. Jennings, E. J. 1953. A rapid test for honey-dew. Textile Industries 117 (12): 120-123.
4. Marsh, P. B., K. Bollenbacher, M. L. Butler, and K. B. Raper. 1949. The fungi concerned in fiber deterioration. II. Their ability to decompose cellulose. Tex. Res. Jour. 19: 462-484.
5. _____, G. V. Merola, K. Bollenbacher, M. L. Butler, and M. Simpson. 1954. The effect of weathering prior to harvest on certain properties of cotton fiber. Plant Dis. Reptr. 38: 106-119.
6. _____, _____, and M. E. Simpson. 1953. Experiments with an alkali swelling-centrifuge test applied to cotton fiber. Tex. Res. Jour. 23: 831-841.
7. Reese, E. T., and M. H. Downing. 1951. Activity of the Aspergilli on cellulose, cellulose derivatives, and wool. Mycologia 43: 16-28.
8. _____, and H. S. Levinson. 1952. A comparative study of the breakdown of cellulose by micro-organisms. Physiologia Plantarum 5: 345-366.
9. Shapovalov, M. 1927. The two most common decays of cotton bolls in the southwestern states. Jour. Agr. Res. 35: 307-312.
10. Siu, R. G. H. 1951. Microbial decomposition of cellulose. 531 pp. Reinhold Publishing Corp. New York City.
11. Thom, Chas., and K. B. Raper. 1945. A manual of the Aspergilli. 373 pp. The Williams and Wilkins Co., Baltimore, Md.
12. White, W. L., R. T. Darby, G. M. Stechert, and K. Sanderson. 1948. Assay of cellulolytic activity of molds isolated from fabrics and related items exposed in the tropics. Mycologia 40: 34-84.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE.

MECHANICS OF THE CONTROL OF HENEQUEN BLACK LEAF SPOT IN EL SALVADOR[1]

Bowen S. Crandall, Leopoldo Abrego, and Bernardo Patino[2]

Black leaf spot of henequen, Agave letonae Trel., caused by Diplodia theobromae (Pat.) Nowell, was first reported by Choussy (1) in 1943. He thought it had probably been present in El Salvador for many years. The disease was described in more detail by Wellman (4) in 1944. Additional studies, 1944-46, were described by Crane and Wellman (2) in 1950. Control by eradication, tried on an experimental scale, failed to show results. By 1947 losses were exceeding 30 percent of the total production and work was resumed in 1948 to bring the disease under control.

The earlier studies accepted the premise, as did the senior writer, that control would have to be by some method of eradication. Locally there was considerable interest in the possibility of control by use of some fungicide or insecticide. A brief study of the economics of the crop demonstrated that a sustained program of this type would probably be out of the question financially.

The henequen plant consists of a rosette of gray-green leaves produced from a central bud at the apex of the stem. As the plant grows the oldest or outermost leaves are harvested. The first harvest occurs at about five years of age. About 10 to 15 leaves are removed per year, in either two or three harvests, usually leaving 20 to 30 leaves on the plant. Harvests continue until the plant matures, flowers, and dies, commonly between the fifteenth and twentieth years.

Because of the plant's manner of growth, eradication of the disease by removing all diseased material, field by field, was out of the question. Even if the cost could be supported the damage to the plants made it impossible. Therefore a method had to be devised to attain the objective during normal harvests and preferably with little or no increase in costs.

The first question raised when eradication was considered was the effect of earlier harvesting of leaves on quantity and quality of fiber. Tensile strength tests on all age leaves showed that fiber quality and quantity were actually progressively better in younger leaves than in old, harvest-age leaves (2).

Perhaps of even greater importance was the second question: What effect would more severe harvests have on the plant and on yields? An experiment was started in 1948 and continued for two years in which plants were defoliated to from 2 to 20 leaves and maintained at that number by quarterly harvests. It was found that the greatest weight of leaf was produced by plants maintained at about 16 leaves. Leaf production continued normal on plants maintained on as few as eight leaves, but weight of leaf declined seriously below 12 to 14 leaves. The results of this defoliation experiment are summarized in Table 1.

Information obtained on the life cycle of the pathogen was immediately useful in control eradication. The infection court was found to be on the unfolding inner leaves, often through insect punctures. The leaf spot developed slowly during the year or more needed for the infected leaf to reach the outside of the plant. Development in size was slow at first but more rapid as the leaf matured. Sporulation of the fungus occurred only on old, dried spots, seldom while the leaf was still on the plant but regularly on diseased leaves discarded in the fields at harvest time. Leaves with smaller spotted areas were usually not discarded but the infected portion was cut off and left in the field (Fig. 1).

The sum total of the information available indicated that an eradication campaign would succeed, if continuous, but that the first results would not be apparent for more than a year. This was explained to the growers and the following recommendations were put into effect:

1. Remove all old diseased leaves and dead plants from the fields, whenever and as soon as possible; usually during cleaning or cultivation operations.
2. Discard no diseased leaf in the field. Since leaf harvesters are paid on a

---

[1] A contribution from the Centro Nacional de Agronomia, Santa Tecla, El Salvador, a technical agricultural organization operated jointly by the Governments of El Salvador and the United States. United States participation in this work was carried out as part of the technical cooperation program (Point IV) in El Salvador administered by the Foreign Operations Administration.

[2] Respectively, Research Adviser, Pathology, United States Operations Mission to Cuba, formerly assigned to the cooperative work in El Salvador; Subchief and Assistant Subchief, Department of Plant Pathology, Centro Nacional de Agronomia.

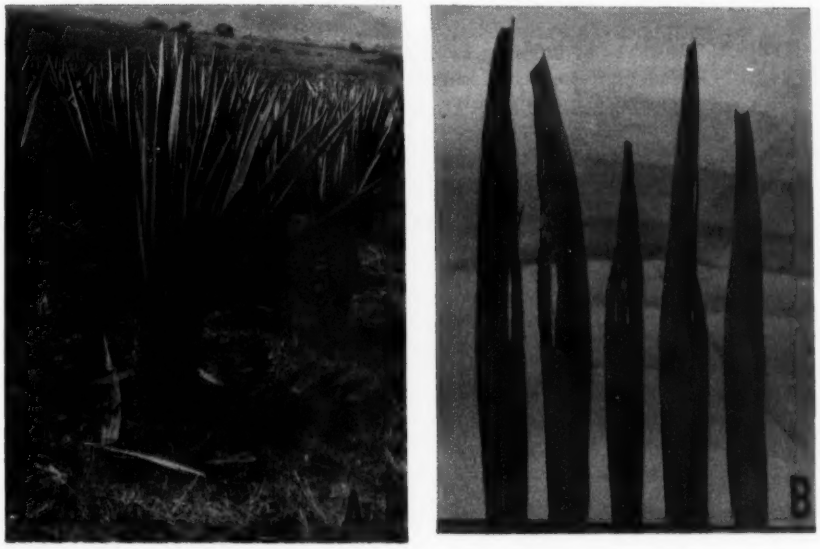


FIGURE 1. A. Spotted leaves discarded in the field during the 1947-48 epiphytotic. B. Leaves with typical black leaf spots.

Table 1. Effect of severity of cut on ten-year old henequen plants on production of leaf during a two-year period.

Number of leaves	Pounds of leaf per plant per month	Number of leaves per plant per month	Average individual leaf weight
2	.23	.97	.237
4	.35	1.25	.280
6	.54	1.32	.409
8	.43	1.40	.307
10	.49	1.49	.329
12	.80	1.42	.563
14	1.05	1.67	.628
16	1.10	1.72	.640
18	1.00	1.51	.662
20	.93	1.57	.592

Table 2. Number of infected leaves, in sample of 240 plants from a typical field, to be found in next three harvests and in residual leaves if cut is regular. January 1950.

Number of leaves per plant	Number of plants	Total number of spotted leaves in: 13th and inner leaves	Leaves 9-12	Leaves 5-8	Outer 4 leaves
12-14	13	1	4	12	28
15-17	40	25	31	58	74
18-20	50	34	47	54	93
21-23	48	43	56	60	84
24-26	40	57	44	47	81
27-29	25	41	29	31	60
30 or more	24	17	10	11	22
Total leaves infected		218	221	273	442
Total leaves		2378	960	960	960
Percent of leaves infected		9.2	23.0	28.4	46.0

Table 3. Number of infected leaves in a sample of 240 plants from a typical field to be found in next three harvests and in residual leaves if the cut is selective. January 1950.

Number of leaves per plant	Number of plants	Total number of spotted leaves in: Inner quartile	Third cut	Second cut	First cut
12-14	13	2	7	11	25
15-17	40	22	35	60	71
18-20	50	19	42	66	101
21-23	48	10	46	81	106
24-26	40	18	30	73	108
27-29	25	13	19	50	79
30 or more	24	7	6	15	32
Total leaves infected		91	185	356	522
Total leaves		1275	1354	1306	1323
Percent of leaves infected		7.1	13.6	27.3	39.5

piece work basis allow the cutters to include diseased leaves in their quotas.

3. Cut a little more severely than usual and always include any diseased leaves encountered next to the whorl cut. This served two purposes: removal of the leaf before the fungus could sporulate and often before the spot reached a size to ruin the leaf.
4. Institute a system of grading out the diseased material at the decorticator and destroy there.

The problem than arose of devising a sampling method to evaulate the results of the work during the period before a measurable decrease in amount of diseased leaf arriving at the decorticator could be seen.

Data on infected leaves could be taken on several hundred plant samples but since the samples varied in the number of leaves contained the question arose as to how to make the quartiles as they would eventually be harvested and arrive at the decorticator. Opinion varied: some thought a regular number of leaves was always taken and a plant once allowed to have 30 leaves always thereafter fluctuated around this number; others thought the cutters judged by color and angle of leaf and that the difference in numbers of leaves indicated variation in rate of plant growth. Perhaps both are correct to some degree. Table 2 (the incorrect method) shows the data evaulated on the basis of a regular cut. Table 3 shows the same data, assuming some criterion on the part of the cutters to allow a greater cut from the more vigorous plants.

Using the sampling method illustrated in Table 3, a typical random sample from a field where control measures had been started in 1949 showed, in September 1950, in the outer quartile being harvested 14.3 percent infection, 17.8 percent in the next quartile, 1.6 percent in the third, and 0.6 percent in the innermost.

In September 1951 samples of 4,000 leaves reaching the decorticator from this same field showed an incidence of disease of 3.1 percent. In 1953 the Minister of Agriculture of El Salvador, in a published interview (3) with a staff correspondent of the Christian Science Monitor on the results of Point IV in El Salvador, stated, "About four years ago a leaf rot blight hit henequen, one of our major export crops. We had no technicians to study it. Point IV went in and in a matter of four months knew what it was, and now it is not a menace. It can be controlled".

Literature Cited

1. Choussy, Felix. 1943. Sobre una enfermedad de origen fungoso en los magueyales de Oriente. El Café de El Salvador 13: 267-269.
2. Crane, Julian C., and Frederick L. Wellman. 1951. Edad del henequén en relacion con las caracteristicas de la fibra. I.I.A.S. Turrialba 1: 74-77.
3. Hallett, Robert M. 1953. Food for El Salvador. Christian Science Monitor for April 30, 1953. (Also reprinted IIAA Building a Better Hemisphere Series No. 27. 1953; and The Havana Post for August 23, 1953).
4. Wellman, Frederick L. 1944. Una mancha negra (Diplodia) de henequén. Sisal de Yucatán 5 (54): 6-7, 30; 5 (55): 6-7, 26.

CENTRO NACIONAL DE AGRONOMIA, SANTA TECLA, EL SALVADOR, A TECHNICAL AGRICULTURAL ORGANIZATION OPERATED JOINTLY BY THE GOVERNMENTS OF EL SALVADOR AND THE UNITED STATES. UNITED STATES PARTICIPATION IN THIS WORK WAS CARRIED OUT AS PART OF THE TECHNICAL COOPERATION PROGRAM (POINT IV) IN EL SALVADOR ADMINISTERED BY THE FOREIGN OPERATIONS ADMINISTRATION.

EAR ROTS IN HYBRID CORN IN ESSEX COUNTY, ONTARIO, IN RELATION TO DAMAGE BY BIRDS[1]

N. J. Whitney[2]

INTRODUCTION

During August of 1952 damage by birds to hybrid corn was evident in various locations in Essex County. Ear rots were prevalent in a large proportion of the damaged ears, whereas those not damaged were nearly free from disease. To determine the relationship between bird damage and ear rots, an investigation was carried out in a number of affected fields in various parts of the county. The results of the investigations are recorded in this paper.

FIELD OBSERVATIONS

Preliminary observations showed that damage was greatest in fields close to bird shelters such as trees and farm buildings, and therefore further observations were concentrated on fields so located. It was soon apparent that the type of injury with which the investigation was concerned was caused primarily by two kinds of birds, namely, the red winged blackbird (Agelaius phoeniceus phoeniceus L.) and the starling (Sturnus vulgaris L.). When the ears were in the milk stage, both species were observed invading corn fields early in the morning. After tearing apart the husks, they exposed the tips of the ears and fed on the soft, starchy part of the kernels. In most cases the seed coat was left attached to the cob (Figures 1 and 2).

In each field the first row selected for ear examination was 15 rows from the side closest to a bird shelter. Subsequent rows were chosen at ten-row intervals towards the centre of the field. In each row 100 consecutive ears were counted and the nature and extent of both bird injury and injury of concomitant types were recorded. Data obtained from counts in ten different fields are included in Tables 1 to 3, inclusive.

Table 1. Extent of bird damage to ears of corn in ten different fields and incidence of disease in damaged and undamaged ears.

Field	Number of ears examined	Ears damaged by birds	Incidence of disease	
			Damged ears	Undamaged ears
		Percent	Percent	Percent
1	200	84.0	18.5	1.0
2	200	83.5	42.5	1.6
3	300	81.3	21.7	0
4	300	78.0	16.2	0
5	500	71.8	29.5	2.1
6	400	65.3	11.5	0
7	200	63.0	11.1	0.8
8	300	57.3	8.7	0.4
9	340	44.1	33.3	3.6
10	300	23.5	31.9	0
Average	304	65.2	22.5	1.0

RESULTS

As Table 1 shows, ears damaged by birds ranged from 23.5 to 84.0 percent, the average

[1] Contribution No. 1390 from the Botany and Plant Pathology Division, Science Service, Department of Agriculture, Ottawa, Canada.

[2] Associate Plant Pathologist.

for the ten fields being 65.2 percent. Individual ears showed a gradation in injury from slight to almost complete destruction of all the kernels (Figure 3). The percentage of damaged ears infected was never less than 8.7 and ranged up to 42.5, the average for the ten fields being 22.5. Undamaged ears in four fields showed no sign of ear rots and in the other six fields ear rot infection did not exceed 8.6 percent.

In Table 2 are recorded the different types and the frequency of occurrence of the ear rots encountered together with a comparison of their incidence on damaged and undamaged ears.

Pink ear rot, caused by Fusarium moniliforme Sheld. (Gibberella fujikuroi (Saw.) Wr.), occurred most frequently (9.8 percent). This rot ranged from one to two isolated spots on an ear to many spots spread over the entire ear. Diplodia zeae (Schw.) Lév. was responsible for the infection in 6.4 percent of the damaged ears. This was followed by Gibberella ear rot caused by Gibberella zeae (Schw.) Petch (3.3 percent), common smut (Ustilago zeae (Beckm.) Ung. (1.5 percent), and Nigrospora ear rot caused by Nigrospora sphaerica (Sacc.) Mason (0.1 percent).

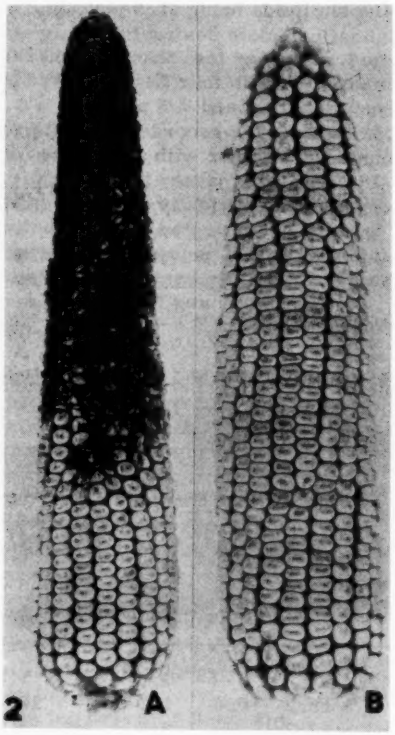
Table 2. Types and percentage incidence of ear rots in bird-damaged and undamaged ears of corn*.

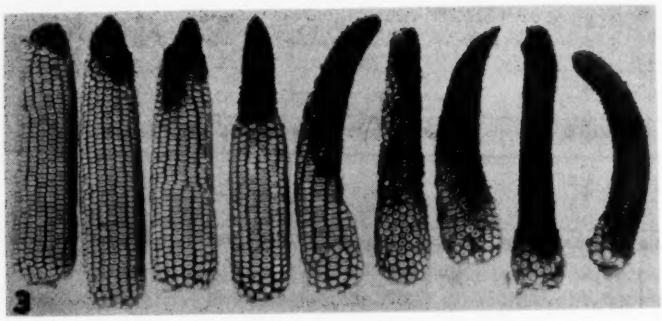
Field	Pathogen											
	Fusarium		Gibberella		Diplodia		Penicillium		Nigrospora		Ustilago zeae	
	Damaged ears	Undamaged ears	Damaged ears	Undamaged ears	Damaged ears	Undamaged ears	Damaged ears	Undamaged ears	Damaged ears	Undamaged ears	Damaged ears	Undamaged ears
1	7.8	---	2.6	---	5.6	---	1.5	---	---	0.8	0.8	---
2	23.2	0.8	1.1	---	18.4	---	1.1	---	0.4	---	0.8	---
3	12.2	---	2.6	---	5.5	---	1.2	---	---	---	2.0	---
4	3.6	---	2.1	---	6.6	---	1.8	---	---	---	1.2	---
5	10.7	0.8	7.8	0.8	2.5	---	1.7	---	0.3	---	5.6	0.8
6	3.7	---	1.9	---	5.0	---	---	---	---	---	1.1	---
7	3.9	0.6	1.3	---	5.8	---	---	---	---	---	0.9	---
8	4.4	---	---	---	0.4	---	1.5	---	---	---	0.8	0.4
9	12.7	2.4	12.0	---	6.7	---	1.3	---	---	---	0.7	---
10	15.7	---	2.0	---	7.5	---	0.7	---	---	---	1.4	---
Average	9.8	0.5	3.3	0.1	6.4	---	1.1	---	0.1	0.1	1.5	0.1

* The data in this table are based on examination of not less than 150 undamaged ears from each field.

Table 3. Percentage of ears infected at the butt, tip, or over the entire ear.

Pathogen	Location of infection		
	Butt	Entire ear	Tip
Fusarium	39.7	46.2	14.1
Gibberella	43.4	50.6	6.0
Diplodia	70.4	28.4	1.2
Penicillium	70.0	10.0	20.0
Smut	54.5	20.5	25.0
Average	55.6	31.1	13.3





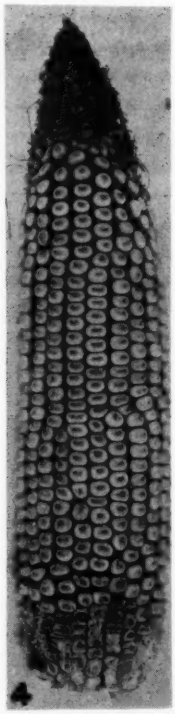


FIGURE 1. A -- damaged ear of corn; B -- undamaged ear. Note the ruptured kernels, caused by the birds, at the tip of the damaged ear.

FIGURE 2. A -- damaged ear; B -- undamaged ear; with the husks removed from both.

FIGURE 3. Damaged ears with the husks removed to show the range of damage from a small amount at the tip of the ear (left) to practically the entire ear (right).

FIGURE 4. An ear of corn slightly damaged by birds, showing a butt infection of Diplodia ear rot.

The incidence of ear rots was negligible in undamaged ears, and it would appear that the protection provided by the husk is an important factor in preventing ear rots, especially those caused by species of Gibberella and Diplodia.

The greatest percentage (55.6 percent) of rotted ears were infected at the butt end (Table 3; Figure 4), whereas only 31.1 percent and 13.3 percent were rotted over the entire ear and at the tip, respectively.

CONCLUSIONS

As regards the incidence of ear rots in Essex County in 1952, this investigation has shown that bird damage is a factor of importance in that it predisposes the corn to infection by ear-rotting fungi. The data show that ear rots were closely correlated with bird damage in the fields investigated. Of 1,640 undamaged ears examined 13 (0.8 percent) were diseased, while out of 2,528 damaged ears 570 (22.5 percent) were found to be diseased. Since there proved to be a much higher incidence of disease in damaged ears than in undamaged ones, and since the relationship between bird damage and disease was very consistent, there is no doubt that the ears were predisposed to infection as a result of the feeding activities of the birds.

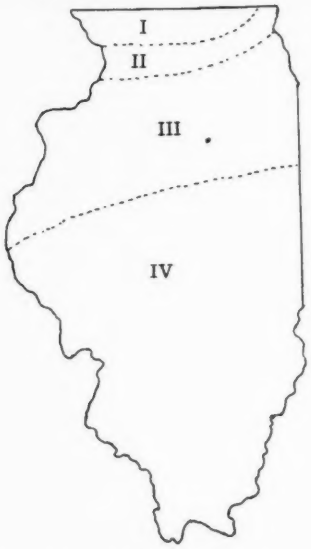
PLANT PATHOLOGY SECTION, SCIENCE SERVICE LABORATORY, HARROW, ONTARIO

STEWART'S DISEASE PROSPECTS IN ILLINOIS FOR 1954

G. H. Boewe

Stewart's disease, or bacterial wilt (Bacterium stewartii) of corn will occur over the entire State and probably will be more destructive in Illinois in 1954 than it was in the summer of 1953.

Two principal types of damage to corn are produced by the disease: an early season wilt, especially in sweet corn, and late season leaf blight, which may be severe enough to cause premature death of plants. The wilt and leaf blight phases of the disease are expected to appear on susceptible corn in Illinois as shown by the table below.



Region	Early Season Wilt	Late Season Leaf Blight
I	Absent or nearly so	Light to moderate
II	Light	Moderate to severe
III	Light to severe	Severe
IV	Destructive	Severe

The four regions listed above are delineated on the map at the left. These regions have been determined on the basis of winter temperature indexes (the sum of the mean temperatures of December, January, and February of the past winter) calculated from the records of 93 weather reporting stations in the State. The data were obtained from the U. S. Weather Bureau office at Springfield. The range of indexes and an average index for each region are shown in the following table.

Region	Range of Indexes	Average Index
I	80.4 - 87.9	84.3
II	86.2 - 89.4	87.5
III	88.4 - 101.6	95.1
IV	98.3 - 123.4	109.6

The chief means of overwintering of Stewart's disease is in the bodies of adult corn flea beetles. These beetles, when they emerge from winter quarters and feed on corn, start the new year's infection. Dr. G. C. Decker, Entomologist of the Natural History Survey, suggests that corn flea beetles can be controlled as follows:

Adult beetles can be controlled by applying DDT at the rate of 1 1/2 pounds per acre, but the control of Stewart's disease through the use of insecticides to eliminate the flea beetles is as yet still definitely in the experimental stage. Available preliminary research data would indicate that five to seven applications of DDT at 1 1/2 pounds per acre, beginning when flea beetles first appear on the seedling plants and at weekly intervals thereafter until beetles are no longer present, will very greatly reduce the incidence of Stewart's disease and such treatments may prove both effective and practical for protecting early plantings of market sweet corn and other plantings of especially high per-acre value.

It seems improbable that field corn and canning corn would justify the adoption of such a program and there are no available data to indicate what modification of the above schedule might prove practical on crops of this type.

Past experience would indicate that in the case of canning corn and large acreages of market corn, avoidance of unwarranted early planting might be very helpful. In 1953, as in previous years, practically complete destruction in sweet corn was confined to plantings made during the first three to seven days of the planting season.

This forecast on Stewart's disease, the sixth to be made for Illinois, is based on the close relationship that appears to exist between the amount of disease which develops during the summer and the temperature of the preceding winter. The development of both phases of the disease, especially on sweet corn, during the past five years coincided very closely with the predictions made in the first five forecasts.

ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA

SEED TREATMENT WITH NEW COMPARED WITH OLDER FUNGICIDES FOR CONTROL OF WHEAT, OAT, AND SORGHUM SMUT IN KANSAS, 1953[1]

E. D. Hansing[2]

Experiments were conducted in the field nursery in 1953 comparing new fungicides with older ones to determine their relative value as seed treatments to control bunt or stinking smut (Tilletia spp.) of wheat, loose and covered smuts (Ustilago avenae and U. kolleri) of oats, and covered kernel smut (Sphacelotheca sorghi) of sorghum. Experiments were conducted also in the nursery to determine the comparative effect of these fungicides on emergence of these crops.

Materials and Methods

The following fungicides were tested in addition to most of those included in experiments from 1950 to 1952 (Hansing 1).

Mergamma -- 3.35% phenyl mercury urea (2% Hg) and 40% lindane, Chipman Chemical Co., Inc., Bound Brook, New Jersey. Applied as a slurry.
MEMA -- 6.35% methoxy ethyl mercury acetate (4% Hg). Chipman Chemical Co., Inc. Applied as a liquid.
MEML -- 3.48% methoxy ethyl mercury lactate (2% Hg). Chipman Chemical Co., Inc. Applied as a liquid.
5025-S -- 25% copper 8-oxy-quinolin. Panogen, Inc., Ringwood, Illinois. Applied as a liquid.
Helexin B -- An antibiotic. C. Leben, University of Wisconsin, Madison, Wisconsin. Applied as a liquid (Dissolved in ethanol).

The methods of inoculation, treatment, storage, and planting were similar to experiments conducted previously (Hansing 1).

Experimental Results

Wheat Bunt -- Highly satisfactory bunt infection was obtained from nontreated seed with from 89 to 94 percent bunted heads occurring for the four dates of planting (Table 1).

Longer storage periods and greater treatment rates gave corresponding decreases in the incidence of bunt for the volatile mercury fungicides, Panogen, Ceresan M, New Improved Ceresan, Agrox, Mergamma, and Setrete. Panogen concentrated and Panogen diluted with water were comparable in the control of bunt. All of the mercury fungicides at their recommended rates, Panogen at 3/4 ounce; Ceresan M, New Improved Ceresan, Agrox, and Setrete, each at 1/2 ounce; and Mergamma at 2 ounces, controlled bunt satisfactorily.

For the nonmercurial fungicides, there was no correlation between storage period and incidence of bunt. When Vancide was increased from 2 to 4 ounces and 5025-S from 3/4 to 1 1/2 ounces, there was a definite decrease in the incidence of bunt. Vancide at 4 or more ounces per bushel was the only nonmercurial to give satisfactory control of bunt.

Oat Smut -- In this experiment the seed was inoculated by the partial vacuum method. Seed not treated produced from 71 to 80 percent smutted heads (Table 1). For the mercury fungicides, both with a longer storage period, and with an increase in rate of treatment, there was a corresponding decrease in smut.

Under the conditions of this experiment, and based on the storage periods of one, two, and four days before planting, the highly volatile mercury fungicides (Panogen and New Improved Ceresan) and MEML were most effective in the control of oat smut. The other volatile mercury fungicides, namely, Ceresan M, Agrox, Setrete, and MEMA, and the nonmercurial fungicide Helexin B, were comparable in the control of this disease. Vancide, Arasan, and Spergon were not satisfactory in the control of oat smut.

Sorghum Kernel Smut -- Good infection was obtained and from 33 to 48 percent smutted heads were produced from seed not treated (Table 1). Seed treated with Panogen at 3/4 ounce, Ceresan M, New Improved Ceresan, Agrox, Setrete (except when planted immediately after

---

[1]Contribution No. 560 and serial No. 438, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan.

[2] Plant Pathologist, Kansas Agricultural Experiment Station, Manhattan.

Table 1. Effect of seed treatment with new compared with older fungicides on control of bunt in Red Chief wheat, smut in Kanota oats, and kernel smut in Red kafir sorghum. Manhattan, Kansas, 1953[a].

Seed Treatment			Days planted after treating										
Fungicide	Form	Rate per bushel	Wheat				Oats				Sorghum		
			0[b]	1	2	4	0	1	2	4	0	1	2
		oz.	Percentage smutted heads										
No treatment	-----	-----	92	94	92	89	76	74	71	80	48	42	33
MERCURIALS													
Panogen conc.	Liquid	1/2	32	23	17	9	--	--	--	--	--	--	--
		3/4	8	3	2	1	38	3	2	1	0	0	0
		1 1/2	3	0	0	T[c]	9	1	T	1	--	--	--
Panogen dil.[d]	Liquid	1/2	27	14	16	6	—	--	--	--	--	--	--
		3/4	9	3	3	0	35	3	3	2	1	0	0
		1 1/2	3	0	0	0	11	2	T	1	--	--	--
Ceresan M	Slurry	1/2	7	3	4	1	42	14	11	5	1	0	0
		1	2	1	1	T	6	2	3	1	--	--	--
N. I. Ceresan	Dust	1/2	6	1	2	T	21	6	4	2	0	0	0
		1	2	1	1	1	4	2	1	0	--	--	--
Agrox	Slurry	1/2	9	4	T	1	34	7	10	7	0	0	0
		1	2	T	0	0	7	2	4	2	--	--	--
Mergamma	Slurry	2	5	4	2	1	29	11	14	11	—	—	—
		4	1	1	T	1	5	4	3	3	--	--	--
Setrete[e]	Liquid	1/2	10	4	1	1	39	16	12	11	2	1	0
		1	3	0	T	0	8	4	6	5	--	--	--
MEMA[e]	Liquid	1/2	--	--	--	--	38	16	15	12	1	0	0
MEML[e]	Liquid	1	--	--	--	--	18	6	3	2	0	0	0
NONMERCURIALS													
Vancide 51	Liquid	2	58	53	61	47	--	--	--	--	2	3	1
		3	23	34	37	19	--	--	--	--	--	--	--
		4	5	8	8	5	--	--	--	--	1	1	0
		5	3	4	5	3	--	--	--	--	--	--	--
		6	2	4	2	1	--	--	--	--	--	--	--
		8	--	--	--	--	22	23	30	43	--	--	--
5025-S	Liquid	3/4	44	42	35	28	—	—	--	--	--	--	--
		1	--	--	--	--	--	--	--	--	1	2	0
		1 1/2	18	22	17	12	--	--	--	--	--	--	--
Helixin B[f]	Liquid	7/10	--	--	--	--	32	10	14	11	--	--	--
		1	--	--	--	--	--	--	--	--	1	0	0
Arasan SF-X	Dust	2	44	47	58	49	--	--	--	--	1	0	1
		6	--	--	--	--	53	58	55	62	—	--	--
Spergon	Dust	2	32	23	27	29	--	--	--	--	--	--	--
		6	--	--	--	--	62	57	54	63	--	--	--

[a] Planted in triplicated 10-foot rows for wheat and oats and 50 foot rows for sorghum, except "no treatment" which was planted in 9 rows for each date of planting. [b] Planted within 5 minutes after treating. [c] T = trace or 0.1 to 0.5 percent. [d] Diluted 1 part Panogen to 4 parts water and applied at 5 times the rate per bushel. [e] Diluted with water so that 4 cc and 8 cc furnished the proper dosage per liter, at 1/2 and 1 ounce per bushel, respectively. [f] Antibiotic dissolved in ethanol.

Table 2. Effect of seed treatment with new compared with older fungicides on emergence of wheat and sorghum planted zero to three years after treating, Manhattan, Kansas, 1953[a].

Seed treatment			Crop and number of years treated					
Fungicide	Form	Rate per bushel	Pawnee and Comanche wheat			Combine and Midland sorghum		
			0	1	2	0	1	2
		ozs.	Percentage emergence					
No treatment	---	---	40	44	9	67	19	24
MERCURIALS								
Panogen conc.	Liquid	1/2	40	47	--	--	--	--
		3/4	44	52	16	79	52	48
		1 1/2	38	43	12	77	--	--
Panogen dil.[b]	Liquid	1/2	41	--	--	--	--	--
		3/4	43	--	--	80	52	--
		1 1/2	39	--	--	--	--	--
Ceresan M	Slurry	1/2	45	42	12	81	47	37
		1	39	33	11	75	--	--
N. I. Ceresan	Dust	1/2	41	38	14	78	46	34
		1	34	31	12	73	--	--
Agrox	Slurry	1/2	41	47	--	78	38	--
		1	33	41	--	76	--	--
Mergamma	Slurry	2	29	--	--	--	--	--
		4	22	--	--	--	--	--
Setrete[c]	Liquid	1/2	40	--	--	77	40	--
		1	28	--	--	--	--	--
MEMA[c]	Liquid	1/2	--	--	--	76	--	--
MEML[c]		1	--	--	--	79	--	--
NONMERCURIALS								
Vancide 51	Liquid	2	42	53	19	78	--	--
		3	45	--	--	--	--	--
		4	47	59	22	81	38	44
		5	46	--	--	--	---	--
		6	47	58	20	--	--	--
5025-S	Liquid	1	49	--	--	77	53	--
Arasan SF-X[d]	Dust	2	48	57	17	81	53	55
Spergon	Dust	2	48	55	18	80	45	48
Significant difference 5%			5.3	6.7	6.2	4.8	6.3	8.5

[a] Each test planted in 5 randomized replications with 200 seeds in each 12-foot row.

[b] Diluted 1 part Panogen to 4 parts water and applied at 5 times the rate per bushel.

[c] Diluted with water so that 4 cc and 8 cc furnished the proper dosage per liter, at 1/2 and 1 ounce per bushel, respectively.

[d] Arasan used on wheat seed treated 1 and 2 years before planting for wheat and 2 years before planting for sorghum.

treating), and MEMA, each at 1/2 ounce, and MEML at 1 ounce produced 1 percent or less smut for all dates of planting.

Seed treated with Vancide at 4 ounces, 5025-S at 1 ounce (two out of three dates of planting), Helixin B at 1 ounce, and Arasan at 2 ounces produced 1 percent or less smutted heads.

Wheat Emergence -- For seed treated and planted two weeks later, significant increases in emergence were obtained with Vancide at 4, 5, and 6 ounces, 5025-S at 1 ounce, and Arasan and Spergon each at 2 ounces per bushel (Table 2). Significant decreases in emergence were obtained with Mergamma at 2 ounces and with New Improved Ceresan, Agrox, Mergamma, and Setrete, each at double their recommended rates.

For wheat planted one and two years after treating, significant increases in emergence were obtained with Panogen at 3/4 ounce, Vancide at 2, 4, and 6 ounces, and Arasan and Spergon, each at 2 ounces. Significant decreases in emergence were obtained for wheat planted one year after treating with Ceresan M and New Improved Ceresan at double their recommended rates.

Sorghum Emergence -- For seed treated and planted two weeks later significant increases in emergence were obtained for all treatments and at all rates per bushel included in the experiment. Increase in rate per bushel decreased emergence for mercury fungicides but increased emergence for Vancide.

For seed planted one and two years after treating, significant increases in emergence were obtained with all of the fungicides tested.

Vancide 51 was recommended in Kansas as a seed treatment for wheat and sorghum (Hansing 2, and Hansing and King 3).

Literature Cited

1. Hansing, E. D. 1953. Seed treatment with new as compared with older fungicides for the control of wheat, oat, and sorghum smut and Victoria blight of oats in Kansas, 1950 to 1952. Plant Dis. Reptr. 37: 49-58.
2. _____. 1954. Cereal and forage crop seed treatment in Kansas. Kansas Certified Seed. Spring 1954: 22-30.
3. _____, and C. L. King. 1953. Vancide 51: A new nonmercurial seed treatment for wheat and sorghum. Kansas Agr. Exp. Sta., Dept. Botany and Plant Pathology. Mimeo. Leaflet No. 7, 1 p., Dec. 17.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, KANSAS STATE COLLEGE, MANHATTAN, KANSAS

FURTHER STUDIES ON THE EFFECT OF CHEMICAL SEED TREATMENTS ON NODULATION OF LEGUMES

L. A. Brinkerhoff, George Fink, R. A. Kortsen, Donald Swift

Several investigators have reported that chemically treated legume seeds produce plants with nodules when grown in soil infested with the effective strains of nodule bacteria. In general the size and number of nodules were not adversely affected except with chemicals like copper sulfate, boric acid, or mercuric chloride (3, 4, 7).

Relatively few tests, however, have been reported on the effectiveness of nodulation in non-infested soil when the nodule-producing bacteria were applied to chemically treated seed. Results of greenhouse tests in sterilized soil are contradictory. Kadow et al. (5) found that Semesan (1% ethyl mercury phosphate), cuprous oxide, and zinc oxide severely reduced nodulation of English peas. On the other hand, Appleman (2) in subsequent tests obtained good nodulation with Semesan of English peas and soybeans, while Ceresan (5% ethyl mercury phosphate) reduced nodulation of peas but not of soybeans. Cuprous oxide prevented nodulation of both peas and soybeans. Kernkamp (6) found that Spergon (tetrachloro-parabenzoquinone) reduced nodulation in steamed soil as compared to non-treated inoculated checks, but Spergon-treated non-inoculated seed produced almost as many nodules as Spergon-treated inoculated seed. It would appear that either the soil was not completely sterilized or became recontaminated. More recently Vlitos and Preston (9) reported that treating inoculated seed with Spergon, Phygon (2, 3 dichloro 1-4 napthoquinone) and Arasan (tetramethyl-thiuram-disulfide) enhanced nodulation of alfalfa and cowpea seedlings in greenhouse tests.

In field tests with soybeans conducted in Iowa, Sherf and Reddy (8) obtained reduced yields when either Arasan or Spergon was used in conjunction with inoculation, provided that the soils did not previously harbor the nodule bacterium. In contradiction, results reported by the Nitragin Co., Inc. (1) from a field test in Wisconsin show very little reduction in nodulation of soybeans when Arasan-treated seeds were inoculated as compared to non-treated inoculated seeds.

Because of many inquiries regarding the compatibility of the nodule bacteria and seed protectant fungicides further studies were initiated at the Oklahoma Station in 1950. Both field and greenhouse tests were undertaken.

FIELD TESTS WITH SOYBEANS

Materials and Methods. Tests were conducted at different locations on the Oklahoma A. & M. College Agronomy Farm at Perkins, Oklahoma, in 1950 and 1951. Check plots indicated that in both years the soil did not previously harbor, to any appreciable degree, the common soybean strain of the nodule bacterium. Previous crops of mung beans, however, had been heavily nodulated, indicating that the cowpea-mung bean strain of Rhizobium was present in the soil.

Seed of the S-100 variety of soybeans were treated several days before planting with Arasan, Spergon, and Phygon at 2 oz. per bu., both as slurry preparations and as dusts. The nodule inoculum, as commercial preparations in ground peat, was applied at 4 oz. per bu. (double the recommended rate) immediately before planting to both dry and moistened seed.

One lot of seed was coated with finely ground talc, after treatment with Arasan as a slurry, in an attempt to reduce possible chemical injury to the nodule inoculum. Another lot of seed was treated with steam sterilized inoculum to determine whether killing the nodule bacteria might have any effect on the protective action of the seed protectant chemicals.

The 1950 test was a randomized block design with four replicates. Individual plots consisted of three rows spaced 24 inches apart and 20 feet long. The planting was made on June 1 with a Planet Jr. seeder and an attempt was made to prevent contamination from plot to plot by rinsing the seed box and seed spout thoroughly with 70% ethyl alcohol between treatments.

The 1951 plantings were made by hand with 100 seeds per row, and contained three-row plots of the same dimensions as 1950. The rows were opened with a garden plow, and covered with a Planet Jr. seeder with openers removed. Two plantings were made on June 2, and an additional two on June 23. The planting design was a Latin square in each instance.

The cowpea strain of inoculum was accidentally used in place of the soybean strain in the two plantings on June 2. One of these tests was made with the Ogden variety of soybeans and the other with S-100. Both plantings on June 23 were with S-100 variety of soybeans and the soybean strain of inoculum. As a precaution against contaminating the seed while planting,

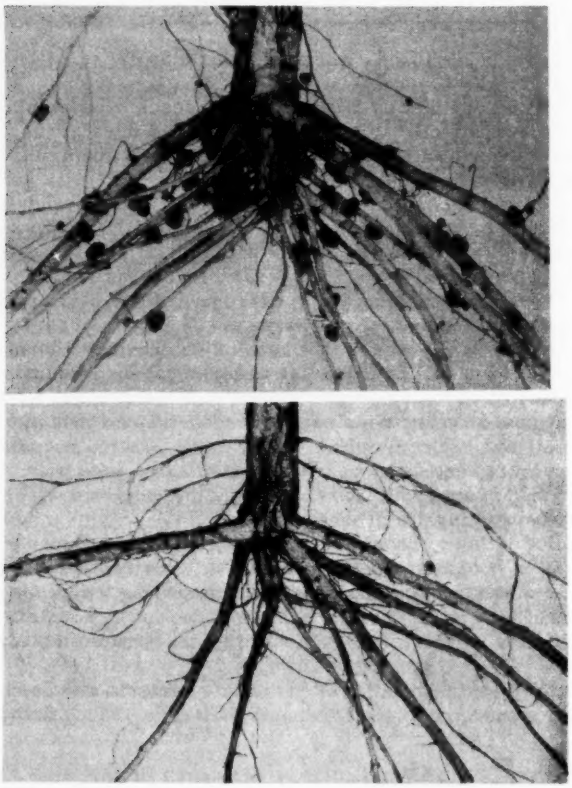


FIGURE 1. Photographs illustrating differences in nodulation of two soybean varieties grown in a field plot in which a previous crop of mung beans had been well nodulated. A, a representative plant of the Ogden variety. B, a representative plant of S-100.

each person carefully rinsed his hands with 70% alcohol after completing a given treatment.

Stand counts were taken after 14 to 21 days, and nodule counts at varying intervals throughout the season. One to three plants were carefully removed from each of three locations in the outside rows of each plot, and the total number of nodules recorded.

Seed yields were obtained only from the center row of each plot. Soil moisture was excellent throughout the growing season in 1950, the rainfall being much above normal during July and August. The 1951 plantings were severely damaged by drought during August and early September.

Results: The number of nodules per plant on four different dates and yields are shown in Table 1 for the 1950 test. Stand counts, nodules per plant, and yields for the two 1951 tests with soybean inoculum are presented in Tables 2 and 3.

The data show that in 1950, a season with abundant soil moisture, Arasan-treated, inoculated seed produced significantly fewer nodules than non-treated inoculated seed. There was somewhat less reduction when dry inoculum was applied to treated seed that were coated with talc, but the difference was not statistically significant at the 5 percent level. Inoculum applied dry to Arasan-treated seed was more effective than inoculum applied wet.

In 1951, a season with a severe summer drought, a much greater reduction in nodulation resulted with chemically treated seed. Dry inoculum was only about one-fourth as effective as wet inoculum on non-treated seed.

The tests with cowpea inoculum demonstrated that the Ogden variety of soybeans was compatible with a different strain or species of Rhizobium than S-100. Nodule counts made on July 27 for all treatments averaged 64.1 for Ogden and only 1.3 for S-100 (see Figure 1). The non-inoculated check for Ogden averaged 65.4 nodules per plant and the inoculated check 75.1; hence it was evident that the effective strain was already present in the soil, and could possibly

Table 1. Effect of seed treatment with Arasan on the average number of nodules per plant and yield of soybeans. Perkins, Oklahoma. 1950.

Treatment	Number of nodules per plant				Yield
	July 11	July 20	July 30	Nov. 2	bu./acre
Arasan dust					
Inoculum (wet)	0.7	3.5	6.6	33.9	28.3
Inoculum (dry)	4.4	8.1	17.2	44.9	30.4
Arasan slurry					
With talc and inoculum (dry)	3.6	17.9	17.4	56.0	31.3
No inoculum	0.1	0.1	0.3	6.9	28.6
No chemical					
Inoculum (wet)	9.3	19.7	32.2	75.1	33.2
No inoculum	0.0	0.2	0.4	2.8	29.9
L.S.D. 5%			12.4	18.5	not sig.

Table 2. Effect of three seed treatment chemicals on stand, average number of nodules per plant and yield of S-100 soybeans. Perkins, Oklahoma, 1951.

Treatment	Stand (Percent of seed planted)	Number of nodules per plant			Yield
		July 23	Aug. 10	Sept. 27	bu./acre
Spergon slurry					
Inoculum (wet)	84	0.6	2.6	4.4	13.2
Arasan dust					
Inoculum (wet)	82	0.7	4.8	3.0	14.0
Phygon slurry					
Inoculum (wet)	76	0.2	2.5	1.3	12.9
No chemical					
Inoculum (wet)	80	11.4	56.4	45.8	13.5
Inoculum (dry)	79	1.0	9.2	10.4	13.8
No inoculum	77	0.1	1.7	3.9	12.7
L.S.D. 5%	4.1	3.3	11.2	8.3	not sig.

Table 3. Effect of Spergon seed treatment on stand, average number of nodules per plant and yield of S-100 soybeans. Perkins, Oklahoma, 1951.

Treatment	Stand (Percent of seed planted)	Number of nodules per plant			Yield bu./acre
		July 23	Aug. 10	Sept. 27	
Spergon slurry					
Inoculum (wet)	83	0.9	11.8	14.7	14.0
Sterilized inoculum (wet)	87	0.1	1.8	2.1	14.4
Inoculum (dry)	86	0.3	3.6	3.2	14.2
No inoculum	83	0.3	1.9	3.5	13.6
No chemical					
Inoculum (wet)	76	13.0	46.2	56.8	13.8
No inoculum	79	0.3	1.2	2.0	13.9
L.S.D. 5%	3.6	2.4	6.4	5.8	not sig.

Table 4. Effect of chemical seed treatments on stand and nodulation of Spanish 18-38 peanuts. Stratford, Oklahoma, 1951.

Treatment	Stand (Percent of seed planted)		Number of nodules per plant	
	Test 1	Test 2	Test 1	Test 2
Phygon slurry				
Inoculum (wet)	59.7	----	46.9	----
Inoculum (dry)	60.4	64.0	58.4	39.7
Sterilized inoculum (wet)	61.5	----	53.9	----
No inoculum	60.4	----	51.6	----
Spergon slurry				
Inoculum (dry)	----	62.0	----	33.9
2% Ceresan				
Inoculum (dry)	----	60.0	----	43.7
No chemical				
Inoculum (dry)	----	50.7	----	38.9
Inoculum (wet)	47.8	39.0	55.3	38.3
No inoculum	48.1	48.1	48.2	36.3
L.S.D. 5%	11.4	13.5	Not sig.	Not sig.

have been the cowpea-mung bean Rhizobium.

Stands were improved by as much as 16 plants per 100 seeds with Spergon and by 12 plants with Arasan in the two tests planted June 2, 1951. Smaller, but statistically significant differences were obtained for the June 23 plantings (see Tables 2 and 3). There was no indication that the addition of either viable or non-viable inoculum to treated seed reduced the effectiveness of the chemicals as seed protectants.

Yield differences were not statistically significant either season. The relatively high fertility level of the soil may have obscured possible benefits from nodulation.

FIELD TESTS WITH PEANUTS

Two tests were conducted with shelled peanut seed at Stratford, Oklahoma in 1951. Various combinations of Phygon and nodule inoculum were used in one 6 x 6 Latin square design, and Spergon, Phygon, and 2% Ceresan with inoculum were compared in a second 6 x 6 Latin square design. Phygon and Ceresan were applied at the rate of 3 ozs. per 100 lbs. of shelled seed and Spergon at 6 ozs. per 100 lbs. of seed. Inoculum in ground peat was applied at 8 ozs. per 100 lbs. of seed. Three-row plots were planted by hand with 75 seeds per row. The planting date was June 22 and the variety was Spanish 18-38. Stand counts were taken after ten days, and nodule counts obtained as described for soybeans. Dry weather reduced plant growth and yields.

Results. Stands and average number of nodules per plant are shown in Table 4. The data indicate that the soil already harbored the peanut nodule bacterium. No significant differences for nodulation were obtained between any of the treatments.

Seed treatments produced significant differences in stands. The addition of inoculum to treated seed did not reduce the effectiveness of the different chemicals.

GREENHOUSE STUDIES

Materials and Methods. Randomized and replicated tests were conducted in 4-inch clay pots of a fine sandy soil that had not been cultivated previously. As a precaution against contamination from drainage water, each pot of soil was placed in the center of a 1-gallon can filled with coarse sand so that the top of the pot was level with the top of the can. The bottoms of the cans were not punctured as the sand provided drainage. The potted soil and cans of sand were sterilized in an autoclave at 15 lbs. pressure for four hours.

For the first three tests the seed were first treated with fungicides as dusts after which the inoculum was applied by the dry method immediately before planting, as recommended by Vlitos and Preston (8). The chemicals were applied at 3 gms. per kg. of seed and the inoculum at approximately 6 gms. per kg. For additional tests the chemicals were thoroughly mixed with the inoculum in the proportion of 1 part chemical to 2 parts of inoculum, and applied to the seed as a slurry.

Chinese Red cowpeas and cowpea inoculum were used for the first three tests and S-100 soybeans and soybean inoculum for the remaining tests. Pots were planted with three seeds each and were later thinned to one plant.

Results. The average number of nodules per plant for the three tests with cowpeas are shown in Tables 5 and 6. In steamed soil Phygon almost completely eliminated nodulation. Spergon, which was used in only one test, averaged 16.0 nodules, compared to 57.0 for the inoculated check. On the other hand, seeds treated with Arasan produced 75.0 nodules per plant compared to 57.0 for the inoculated check. However, the nodules were extremely small for the Arasan treatments in steamed soil. Five of the eight non-inoculated checks in autoclaved soil averaged from 0.5 to 15.5 nodules per plant, indicating that either the soil was not thoroughly sterilized or that the pots had become contaminated during the test. Further tests indicated that contamination was probably responsible for nodulation in non-inoculated steamed soil.

The non-inoculated checks in non-sterilized soil averaged approximately as many nodules as those that were inoculated, indicating that the soil was naturally infested with the cowpea Rhizobium. Chemical treatments did not appreciably affect nodulation in infested soil with the possible exception of Phygon.

In sterilized soil with inoculum applied to the seed most of the nodules were produced on the lateral roots, while in naturally infested soil approximately 1/4 to 1/2 of the nodules

Table 5. Effect of seed treatment with Phygon on nodulation of Chinese Red cowpeas. Greenhouse, 1949-50.

Treatment	Number of nodules per plant	
	After 38 days Test 1	After 28 days Test 2
Sterilized soil		
Inoculum only (dry)	40.0	25.5
Phygon + inoculum (dry)	1.5	2.5
Phygon only	15.5	0.5
No chemical or inoculum	8.5	0.0
Non-sterilized soil		
Inoculum only (dry)	24.0	23.8
Phygon + inoculum (dry)	9.5	19.0
Phygon only	14.0	24.0
No chemical or inoculum	19.0	27.0

occurred on the tap roots. Similar observations have been reported by other workers (2, 8).

In two additional tests in which the nodule inoculum was mixed with 12 different seed treatment fungicides and applied to the seed as a slurry, Vancide 51 (sodium salts of dimethyl dithiocarbamic acid and 2-mercaptobenzothiazole), Ortho 406 (N-trichloromethylmercapto-4 cyclohexene - 1, 2 - dicarboximide), and C 5-3 (supplied by Gallawhur Chemical Corporation) appeared to be the least toxic to the nodule inoculum. However, the results were not consistent between replicates, or for the two tests.

DISCUSSION

The results obtained in the preceding studies indicated that Phygon, Spergon, and Arasan were definitely injurious to the nodule bacteria when chemical and inoculum were applied to the seed. These findings are in agreement with laboratory studies reported by the Nitragin Co. (1), in which a marked decrease in the number of living bacteria was obtained when inoculant-fungicide mixtures were incubated in moist sand and subsequently cultured on agar. Further evidence of toxicity was also reported (1) when paper discs saturated with the chemicals prevented growth of Rizobium on agar plates in zones in which the chemicals had diffused into the agar.

The comparatively good nodulation obtained in the field with Arasan-treated seed by the Nitragin Co. (1) was attributed to avoiding excessive injury by planting immediately after inoculating, increasing the dosage of inoculum, and using the wet method of inoculation.

From the present Oklahoma studies it would appear that the initial number of nodules produced on young plants and the relative abundance of soil moisture may be important factors in subsequent nodulation during the season. A marked reduction in nodulation with treated-inoculated seed was evident four to six weeks after planting in both the field and greenhouse tests. Young plants with 1 to 4 nodules became reasonably well nodulated in the field in 1950 but this was not true in 1951 when a severe drought occurred during the middle part of the growing season. However, plants that averaged 11 and 13 nodules when four weeks old became reasonably well nodulated in 1951. If nodulation of the new roots is dependent upon the dispersal of inoculum from primary nodules, this might then account for the erratic results obtained with treated-inoculated seed.

High soil moisture was suggested (1) as a factor which aided the bacteria to migrate away from the treated zone. The development of nodules on lateral roots supports the hypothesis that migration occurs; however, it would appear that migration could occur at the time of planting or possibly after the protective chemicals had been rendered non-toxic. Appleman (2) obtained viable nodule bacteria from seed that had been treated with 5% Ceresan or cuprous oxide for 24 hours, but only after thoroughly rinsing the treated seed with water. He suggested that either the chemical action was bacteriostatic or that the bacteria had been protected by migrating into crevices in the seed coat.

Table 6. The effect of seed treatment with Arasan, Phygon and Spergon on nodulation of Chinese Red cowpeas. Greenhouse test January 24 to March 3, 1951.

No.	Treatment	Number of nodules per plant (after 38 days)		
		On tap root	On laterals	Total
	Sterilized soil			
1.	Inoculum only (dry)	1.5	55.5	57.0
2.	Arasan + inoculum (dry)	0.0	75.0	75.0[a]
3.	Phygon + inoculum (dry)	0.5	2.5	2.5
4.	Spergon + inoculum (dry)	0.5	16.0	16.0
5.	Arasan only	0.0	0.0	0.0
6.	Phygon only	0.0	0.5	0.5
7.	Spergon only	0.0	0.0	0.0
8.	No chemical or inoculum	0.0	6.0	6.0
9.	Non-sterilized soil			
1.	Inoculum only (dry)	7.0	12.5	19.5
2.	Arasan + inoculum (dry)	5.0	10.5	15.5
3.	Phygon + inoculum (dry)	4.0	5.5	9.5
4.	Spergon + inoculum (dry)	12.0	26.0	38.0
5.	Arasan only	6.5	14.0	20.5
6.	Phygon only	4.0	16.5	20.5
7.	Spergon only	6.5	30.0	36.5
8.	No chemical or inoculum	6.5	16.5	23.0

[a] These nodules were all very small.

Doubling the recommended dosage of inoculum did not insure nodulation in the Oklahoma tests. However, increasing the amount of inoculum did not appear to affect the protective value of the chemicals.

Coating treated seed with talc before inoculating resulted in slightly more nodules in one test with soybeans. Kadow et al. (5) obtained an increase in nodules when graphite was used with cuprous oxide.

These studies emphasize the complexity of the inoculant-seed protectant problem and indicate the need for further research.

Summary

Phygon, Spergon, and Arasan tended to reduce or in some instances nearly eliminate nodulation in non-infested soil when soybeans and cowpeas were grown from treated, inoculated seed. In the field tests, the reduction was more pronounced in 1951 when the plants suffered from severe drought in August. The dry method of inoculation was not effective regardless of chemical treatment in 1951.

No evidence was obtained that the addition of nodule inoculum to the seed decreased the protective value of the seed treatment chemicals. In one test with shelled peanuts, wet inoculum without a seed protectant reduced the stand.

The Ogden variety of soybeans was well nodulated, apparently by the cowpea-mung bean Rhizobium, but the S-100 variety was clearly not compatible with the same strain.

Nodulation was not appreciably affected by seed treatments in naturally infested soil.

Literature Cited

1. Anon. 1952. Seed treatments and inoculation. The Soybean Digest 12: 14-16. (Contributed by Research Department, The Nitragin Co., Inc.).
2. Appleman, M. D. 1941. Effect of seed treatment on nodulation of soybeans and peas. Proc. Soil Sci. Soc. Amer. 6: 200-203.
3. Buchholtz, W. F. 1936. Seed treatment as a control for damping off of alfalfa and other legumes. Phytopath. (Abstr.) 26: 88.
4. Duggar, J. F. 1935. Nodulation of peanut plants as affected by variety, shelling of seed, and disinfection of seed. Jour. Amer. Soc. Agron. 27: 286-288.
5. Kadow, K. J., L. E. Allison, and H. W. Anderson. 1937. Effect of chemical treatment of pea seed on nodulation by Rhizobium leguminosarum. Illinois Agr. Exp. Sta. Bul. 433: 3-12.
6. Kernkamp, M. F. 1948. Chemical treatment of soybean seed in relation to nodulation by nodule bacteria. Phytopath. 38: 955-959.
7. Litynski, A. 1937. Influence of seed corrosive agents on the development of root nodules on bush beans. Polish For. Ann. 38: 343-365. (Chem. Abstr. 31: 6803. 1937).
8. Sherf, A. F., and C. S. Reddy. 1952. Seed treatment and inoculation studies on soybeans in Iowa fields, 1950-1951. (Abstr.). Phytopath. 42: 19.
9. Vlitos, A. J., and D. A. Preston. 1949. Seed treatment of field legumes. Phytopath. 39: 706-714.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND DEPARTMENTS OF BOTANY AND PLANT PATHOLOGY, AND AGRONOMY, OKLAHOMA A & M COLLEGE, STILLWATER, OKLAHOMA

AN EXPERIMENTAL BLADE-TYPE SOIL FUMIGANT APPLICATOR

Harold J. Jensen and Glen E. Page[1]

Liquid nematocides have been applied to the soil for the control of soil-borne plant parasitic nematodes for several years. Nearly all large scale treatments are applied with power-drawn equipment which usually consists of some type of plow-sole or chisel applicator. There are many disadvantages to overcome in present equipment although practical control is being achieved in many instances. Results with chisel applicators are frequently unsatisfactory under Oregon conditions. The effectiveness of treatment with this type of equipment is dependent on a continuous flow of the material through the applicator tubes into the chisel furrows and the subsequent diffusion into areas between the chisel paths. Frequently the applicator tubes become plugged, leaving untreated areas in the field. Plow-sole application is an extremely arduous task due to the difficulty encountered when attempting to plow Oregon soils that are in the recommended seed-bed condition.

The Departments of Agricultural Engineering and Botany & Plant Pathology of Oregon State College have developed an experimental blade-type applicator. The applicator (Fig. 1) is designed to apply the nematocide in a continuous sheet at the 6- or 7-inch injection depth, which is accomplished by forcing the material under pressure through a spray-boom occupying a protected recess beneath the blade (Fig. 2). The nematocide is forced through the spray system by a gear-driven pump attached to the power take-off of the tractor. The experimental

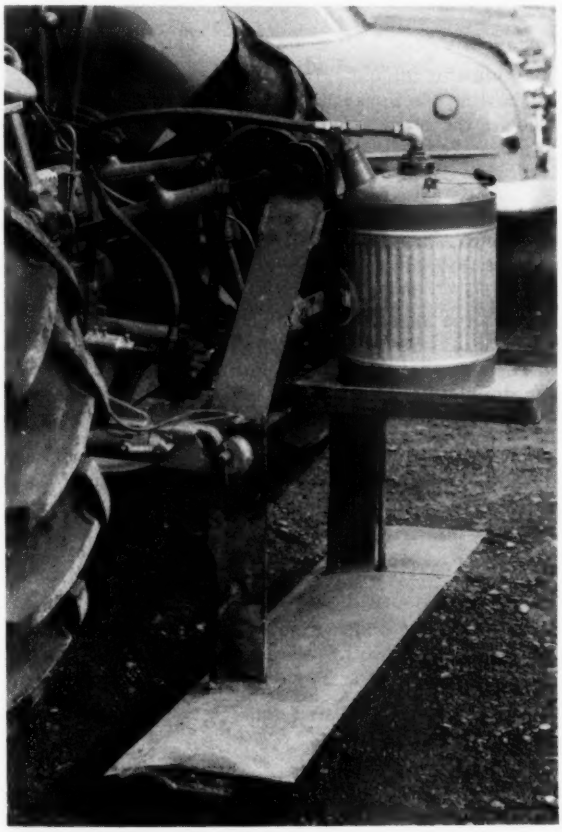


FIGURE 1. A side view of the experimental blade-type applicator developed by the Departments of Agricultural Engineering and Botany & Plant Pathology at the Oregon Experiment Station. (Photograph by H. H. Millsap).

[1]Assistant Nematologist, and Assistant Agricultural Engineer, respectively, Oregon Agricultural Experiment Station.

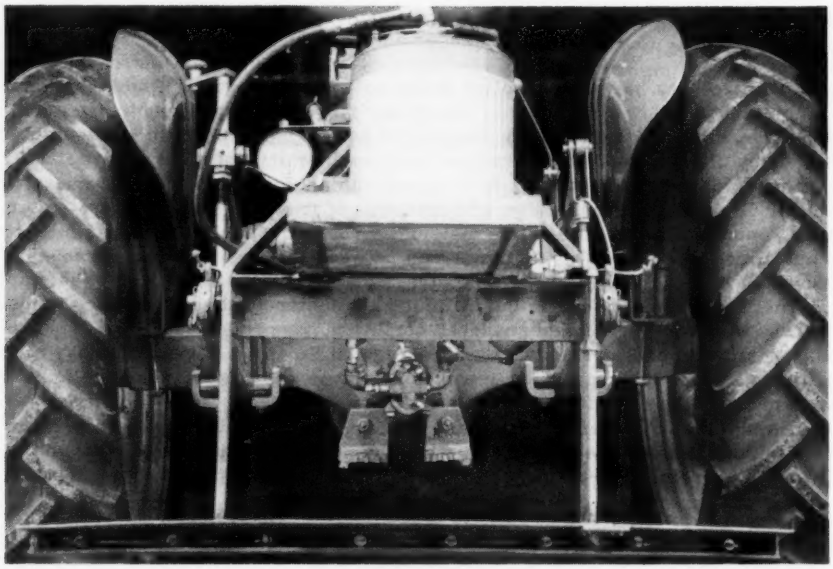


FIGURE 2. A rear view of the experimental blade-type applicator showing the spray boom occupying a protected recess beneath the blade. (Photograph by H. H. Millsap).

blade-type applicator is designed to fit tractors having a three-point hitch. No difficulty was experienced in treating soils that were relatively free of large clods and unrotted straw and other plant materials.

Data comparing the control efficiency of the experimental blade-type applicator with that of conventional equipment will be published later. It is hoped that with this equipment a more complete nematode kill can be achieved resulting in a longer period between treatments than that obtained with the conventional applicators. The plans of the experimental blade-type applicator will be available when additional tests have been completed.

DEPARTMENTS OF BOTANY & PLANT PATHOLOGY AND AGRICULTURAL ENGINEERING, OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON

A FOLIAGE DISEASE OF POTATO INDUCED BY CHAETOMIUM SPECIES[1]

Allyn A. Cook

During the course of routine greenhouse studies in 1951-52, black necrotic lesions were found on the leaves of several potato plants of the varieties Cherokee and Bliss Triumph. These lesions were similar to those of early blight (Alternaria solani) except that concentric ridges were absent. Two series of isolations from these diseased leaves resulted in cultures of four Chaetomium species, tentatively identified as C. aureum, C. globosum, C. torulosum, and C. trilaterale[2,3]. Since no report could be found of species within this genus producing a potato disease, a series of preliminary experiments were conducted to determine the disease producing capabilities of these species. C. tortile, previously isolated from tubers, was also included in these experiments.

Materials and Methods: The five species of Chaetomium were isolated from the original diseased leaves or tubers on potato-dextrose-agar (2%). Chaetomium aureum, C. globosum, and C. trilaterale fruited abundantly on this medium but C. tortile and C. torulosum produced no perithecia. The latter two species also failed to produce perithecia on various other media, including water agar, water agar with 10 g./l. dextrose, Leonian's medium, malt agar, and an inulin medium[4]. Sparse amounts of perithecia were finally formed in old cultures on potato-dextrose-agar as these dried at room temperature.

The disease producing capabilities of these fungi were tested on plants 6 to 10 inches high on which the leaves were either injured or uninjured. The injuries were made just prior to inoculation by punching holes into leaves with straight pins set in a rubber stopper. Inocula of the fungi were prepared from cultures on potato-dextrose-agar in petri dishes by suspending the surface growths in sterile, distilled water and dispersing these in a Waring Blendor for about one minute. These were applied to the leaves from an atomizer, a sterile pipette or a transfer loop. Immediately following inoculation the plants were placed in a moist chamber for 48 hours before being returned to a greenhouse bench.

Results: Leaf injuries were necessary for symptom expression. Necrosis was manifest at the periphery of the injuries within seven days after inoculation. Thereafter the necrotic areas increased in size and either remained discrete or coalesced with others in their immediate vicinity (Figure 1). The lesions did not increase further in size after three weeks. At this time chlorosis usually was apparent around severely affected areas. The fungi could be reisolated readily from the diseased areas. No symptoms were produced on uninoculated, injured check plants.

Toxin Formation: As necrosis may result from toxins formed in culture or on the leaf by these fungi, the presence of such toxins was tested in the following manner. Suspended inocula, prepared from potato-dextrose-agar cultures as above, were filtered through #30 filter paper and the filtrates put in test tubes. Excised potato plants were placed in these filtrates. Check plants, in tap water, remained turgid, green, and apparently unaffected for as long as 96 hours, while plants in the filtrates usually wilted within 48 hours, and after 10 days exhibited various degrees of intercostal chlorosis and necrosis, and marginal burning.

Ascospore Germinability: Ascospores of all above species failed to germinate on inoculated leaves or in prepared inocula held for as long as ten days. The only source of growth in the prepared inocula was from mycelial fragments. Ascospores also failed to germinate in

---

[1] Contribution from the Plant Pathology Department, South Dakota Agricultural Experiment Station, State College Station, South Dakota. Approved for publication by the Director, as Journal Series No. 313.

[2] Chivers, A. H. 1915. A monograph of the genera Chaetomium and Ascotrichia. Torrey Bot. Club Mem. 14: 155-240.

[3] Skolko, A. J., and J. W. Groves. 1953. Notes on seed-borne fungi. VII. Chaetomium. Can. Jour. Bot. 31: 779-809.

[4] Tschudy, R. H. 1937. Experimental morphology of some species of Chaetomium. Use of cultural reaction in determining species characteristics. Amer. Journ. Bot. 24: 472.

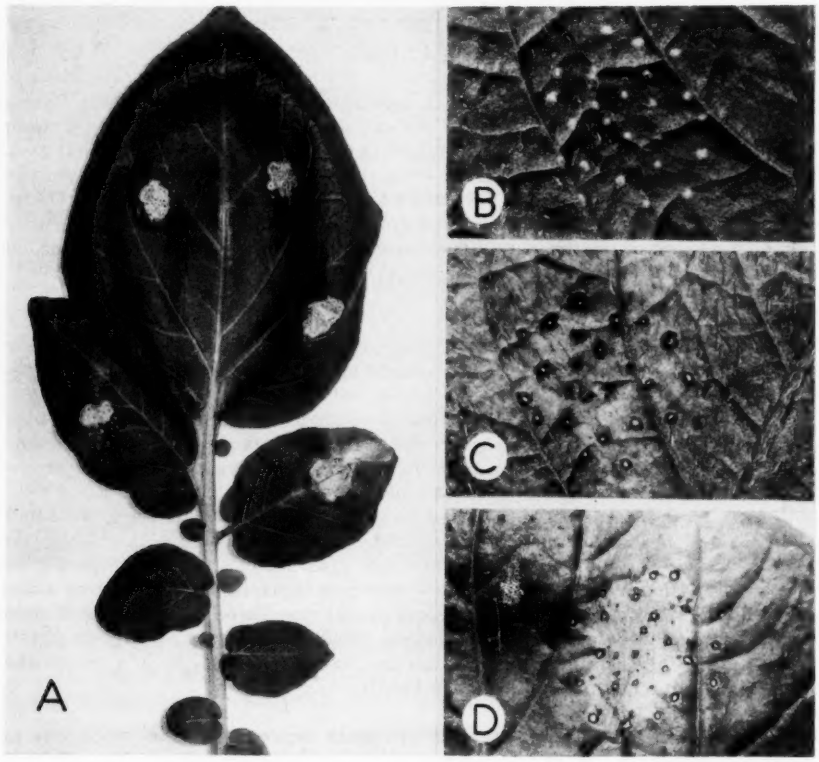


FIGURE 1. Types of symptoms expressed 14 days after inoculation of injured potato leaves with inoculum of:
A) Chaetomium globosum; B) Uninoculated check:
C) C. aureum; D) C. tortile.

aqueous solutions of 0.01, 0.1, and 1.0% ammonium chloride, ammonium nitrate, ammonium sulfate, dextrose, magnesium sulfate, sodium chloride, sodium phosphate, urea, "Dreft", and oxalic acid; 0.5, 1.0, and 2.0% inulin; and 3, 5, and 10% potato leaf decoction. A small percentage of ascospores germinated, however, in 0.1% preparations of ammonium oxalate, sodium biphosphate and yeast extract, and in 1.0% solutions of lactose and potassium phosphate, particularly the latter. Inoculum of C. globosum, prepared with a 1.0% potassium phosphate solution, produced the same symptoms as inoculum with distilled water.

Disease symptoms comparable to those from which the original isolations were made were never obtained. The results of these preliminary experiments indicate, however, that the species of Chaetomium herein tested are capable of inducing a foliage disease of potatoes, either by indirect infection or by production of toxic materials. Further work is necessary for a full understanding of the disease producing capabilities of these fungi.

SOUTH DAKOTA AGRICULTURAL EXPERIMENT STATION, STATE COLLEGE STATION

CONTROL OF BACTERIAL SPOT OF TOMATO AND PEPPER SEEDLINGS WITH AGRIMYCIN[1]

Robert A. Conover

Bacterial spot (Xanthomonas vesicatoria (Doidge) Dowson) has been a serious disease of tomatoes and peppers in the vegetable-growing areas along the Florida Lower East Coast for many years. It is especially destructive during September and October, principally because frequent heavy rains fall during these months. Seedlings in plantbeds, prepared for the fall and winter crops, are especially hard-hit, many of them being rendered unfit for use as transplants. Others, less severely damaged, are set in the field even though considerable infection is present. If dry, sunny weather ensues, little damage occurs in the field; however, the disease may flare up and cause considerable loss when driving rain occurs.

Up to the present time there has been no effective control of bacterial spot in the area. Locating plantbeds on land not previously used for agricultural purposes and the frequent use of copper and/or dithiocarbamate sprays and dusts have not controlled the disease under commercial conditions. In experiments, no fungicide tested has controlled the disease when conditions favored its rapid spread.

Experiments were started in 1952 to determine whether certain antibiotics would control bacterial spot of tomato and pepper seedlings. Pure streptomycin sulfate and oxytetracycline hydrochloride[2] provided by Chas. Pfizer & Company, Inc., were used in these studies. The tests were made on seedlings growing in flats. The plants were inoculated after treatment by watersoaking the leaves with a heavy suspension of the causative organism. Under the severe disease conditions of the experiments, sprays of 0.05% and 0.1% streptomycin sulfate applied three days before inoculation gave excellent control of bacterial spot on both tomatoes and peppers. Higher concentrations caused injury, sprays containing 1% streptomycin sulfate being lethal. Because of difficulty experienced with solutions, results with Terramycin were less conclusive but it was clear that concentrations sufficient to inhibit disease development were rather phytotoxic.

The favorable results with streptomycin were so encouraging that a large-scale experiment was conducted with a commercial grower in the fall of 1953 to determine whether sprays prepared with formulations of crude streptomycin would control bacterial spot in commercial tomato plantbeds. The results of this experiment and of a small-scale experiment on pepper seedlings are reported in this paper.

METHODS, MATERIALS, AND CONDITIONS OF THE EXPERIMENT WITH TOMATOES

Sixty plantbeds, each about 4 feet wide and 500 feet long, were used in the experiment on tomatoes. These were in two blocks of 30 parallel beds, and beds of the two blocks joined end to end although separated by a field road about 12 feet wide. The grower performed all operations on the plantbeds, including spray applications, according to his usual practice. Spray applications were made with a tractor-drawn power sprayer provided with booms which covered five beds. Seven nozzles were used per bed. The grower calculated that spray applications averaged about 100 gallons per acre. Coverage was uniform and generally good according to commercial standards. Manzate was used on a 5- to 7-day schedule for control of fungus diseases and parathion and chlordane were used as required to control insect pests.

An Agrimycin[3] formulation containing 22.2% streptomycin and 2.2% Terramycin was used to prepare sprays containing 100 ppm. and 200 ppm. streptomycin and proportionate amounts of Terramycin. These were applied by the grower. Agrimycin was not mixed with any other material.

The bed straddled by the sprayer was left unsprayed (by plugging the nozzles over this bed) to serve as a check. Thus in each block of 30 beds, 12 were sprayed with 100 ppm. streptomycin and 10 ppm. Terramycin, 12 with 200 ppm. streptomycin and 20 ppm. Terramycin, and six were left unsprayed.

It was intended to make applications four days apart, but on two occasions after hard

---

[1] Florida Agricultural Experiment Station Journal Series, No. 269.

[2] Chas. Pfizer & Co., Inc., antibiotic Terramycin (R).

[3] Chas. Pfizer & Co., Inc., trademark for antibiotic formulation.

rains, applications were made two days apart. Treatments were applied as indicated on September 22, 26 and 28, but, at the insistence of the grower who quickly recognized the control obtained and the hazard of untreated beds, applications on October 3 and 5 were made to all beds, including the checks. At the time of the first application most of the plants in block I had two expanded leaves and the third leaf was emerging; in block II, the first two true leaves were about one-half expanded on September 22.

Bacterial spot was first noted in adjacent plantbeds not in the experiment on September 19. Although it was not seen in block I prior to the first application, it seems certain that some infection was present because the disease was apparent in the check beds on September 26 when the second application was made. On the other hand, infection was not observed until October in the younger plants in block II; this suggests that the first application in this block was made before the disease had become established. High humidity and almost daily rains, totalling approximately 10 inches, which fell during the period between September 22 and October 9, provided favorable conditions for the development of bacterial spot.

The incidence of bacterial spot was measured when the seedlings were 6 to 8 inches tall and ready to transplant to the field. This was done in block I on October 3 by counting the numbers of infection centers in each bed and estimating their area. Infection centers, which were readily apparent to one walking alongside the beds, were manifested by severe spotting and yellowing of the leaflets and epinasty of the leaves. It is recognized that this method of tabulating results did not measure the actual amount of disease present, since light infections not sufficient to cause yellowing or epinasty were ignored. However, the method did afford a means of quickly and uniformly assessing the incidence over a large area and the results obtained are considered an accurate reflection of the control effected with Agrimycin sprays.

Results were recorded in block II on October 9 and 10. Three 25-plant samples were pulled at random at locations 20, 25 and 30 feet from the end of each bed adjacent to block I. Each plant was placed into one of the four following classes: 1) no disease present; 2) a few spots on one or two bottom leaves, but not enough to cause yellowing or epinasty; 3) lesions on three or more leaves with curling, yellowing, or epinasty on two bottom leaves only; 4) lesions on nearly all leaflets with many leaves showing epinasty or yellowing, or dehisced.

RESULTS

Bacterial spot appeared in block I as infections scattered throughout the beds. It was readily apparent that both treatments reduced the amount of bacterial spot in comparison with the amount on the checks, both in number of infection centers and in their size (Table 1). In beds treated with the lower dosage of Agrimycin, an average of 3.4 infection centers per bed was found, each having an average area of 6.35 sq. ft.; the checks among these beds averaged 12 infection centers, each having an average area of 7.71 sq. ft. In beds sprayed with 200 ppm. streptomycin and 20 ppm. Terramycin, an average of only 2.3 infection centers per bed was noted, each averaging 4.09 sq. ft. in area, whereas the checks in this group had an average of 14.3 infection centers, each with an average area of 13.90 sq. ft.

In block II bacterial spot was first observed along the ends of the beds nearest block I, and spread into these beds was largely from this end. However, some scattered infections were also present, but not in sufficient numbers to justify their use as indicators of effectiveness of treatment as was done in block I. Effects of treatments on bacterial spot development in block II are summarized in Table 2. Both dosages of Agrimycin sprays markedly reduced the severity of the disease. Sprays containing 200 ppm. streptomycin and 20 ppm. Terramycin were most effective -- 74 percent of the seedlings being disease-free and only 0.4 percent being severely diseased. Efficacy of the treatments is further indicated by considering the seedlings in terms of transplanting stock. Plants in Class I and II are "good" plants, the disease present in the latter class not being sufficient to affect survival during transplanting. Plants in Class III have little value as transplants and those in Class IV are useless for this purpose. Thus considered, 95 percent of the seedlings sprayed with 200 ppm. streptomycin and 20 ppm. Terramycin were suitable for transplanting; this is to be compared with 73 percent usable from beds sprayed with the 100-10 ppm. dosage and an average of 27 percent usable from the beds of untreated plants.

None of the treatments caused visible injury to the seedlings.

EXPERIMENT WITH PEPPERS

Agrimycin sprays of three concentrations were compared with an untreated check in a

Table 1. Control of bacterial spot in tomato plantbeds in block I with Agrimycin sprays.

Treated beds sprayed with 100 ppm. streptomycin and 10 ppm. Terramycin			Treated beds sprayed with 200 ppm. streptomycin and 20 ppm. Terramycin		
Bed no. (west to east)	No. infection centers	Estimated area (sq. ft.) of bed severely diseased	Bed no. (west to east)	No. infection centers	Estimated area (sq. ft.) of bed severely diseased
1. Treated	---*	---*	16. Treated	5	12
2. Treated	---*	---*	17. Treated	6	56
3. Check	---*	---*	18. Check	15	483
4. Treated	2	9	19. Treated	5	24
5. Treated	5	12	20. Treated	1	1
6. Treated	5	28	21. Treated	0	0
7. Treated	3	19	22. Treated	0	0
8. Check	12	69	23. Check	19	93
9. Treated	3	19	24. Treated	2	2
10. Treated	2	18	25. Treated	3	3
11. Treated	2	21	26. Treated	2	9
12. Treated	1	3	27. Treated	1	1
13. Check	12	116	28. Check	9	20
14. Treated	3	29	29. Treated	2	4
15. Treated	8	58	30. Treated	1	1
Avg. of treated beds	3.4	21.6		2.3	9.4
Avg. of check beds	12.0	92.5		14.3	198.7

*Plants were pulled from these beds before records could be taken.

randomized, replicated experiment for the control of bacterial spot on pepper seedlings. Treatment plots were 2.5 feet wide and 6 feet long. Sprays were applied with a compressed air sprayer in volume sufficient to cause run-off on some leaves. The first application was made on September 26 when most of the seedlings had three leaves; additional applications were made on September 29, October 5, and October 9. Bacterial spot was observed in the plots on September 25, one day before the first spray was applied. Subsequent development of the disease was moderately severe. On October 15, fifteen plants were pulled at random from each plot and the number of diseased and bacterial spot-free leaves counted. Dehisced leaves were counted as being diseased.

The results of this experiment, which are summarized in Table 3, show that Agrimycin sprays effectively reduced the amount of bacterial spot on pepper seedlings. The control obtained from sprays containing 300 ppm. streptomycin and 30 ppm. Terramycin was nearly equal to that obtained with the 800-80 ppm. concentration and was significantly superior to the 100-10 ppm. dosage.

DISCUSSION

Observations suggest that there is no optimum concentration, such as is commonly used with fungicides, of Agrimycin in sprays for bacterial spot control. Rather, the amount required in a given instance depends on the circumstances, more Agrimycin being required in situations

Table 2. Control of bacterial spot in tomato plantbeds in block II with Agrimycin sprays.

Treatment	No. plant-beds	No. plants examined	Average percentage of plants in Class I	Class II	Class III	Class IV
100 ppm. streptomycin and 10 ppm. Terramycin	12	900	47.3	25.7	15.6	11.2
Check (among beds receiving above treatment)	3	225	10.2	11.6	18.2	60.0
200 ppm. streptomycin and 20 ppm. Terramycin	12	900	74.0	21.2	4.4	0.4
Check (among beds receiving above treatment)	3	225	12.8	20.0	32.4	34.8

Table 3. Control of bacterial spot in pepper plantbeds with Agrimycin sprays.

Treatment	Average No. healthy leaves per seedling	Average No. diseased leaves* per seedling
Check	0.60	4.22
100 ppm. streptomycin, 10 ppm. Terramycin	2.25	2.95
300 ppm. streptomycin, 30 ppm. Terramycin	3.62	1.97
800 ppm. streptomycin, 80 ppm. Terramycin	3.85	1.77
Least significant difference at 5% level	0.78	0.78
Least significant difference at 1% level	1.12	1.12

*Includes dehisced leaves

especially conducive to heavy infection and rapid disease development. Aside from its effect on disease development, rainfall occurring within a few hours after treatment appears to lessen the effectiveness of Agrimycin sprays, presumably by rinsing off the antibiotics before maximum absorption has occurred. However, hard rains occurring 24 hours or later after spraying have not appeared to lessen the efficacy of a treatment.

In the large-scale experiment reported herein, excellent control of bacterial spot on tomatoes was obtained with Agrimycin sprays containing 200 ppm. streptomycin and 20 ppm. Terramycin. In several preliminary experiments two to three times as much Agrimycin was required to give control equal to the aforementioned. Observations indicate that the differences in these instances were due to experimental design or technique rather than differences in disease intensity. It appeared that the plots (4 ft. x 6 ft.) used in early experiments were too small, and the effects of adjacent untreated or ineffectively treated plots too great, to permit such experiments to accurately foretell the performance of a given dosage when applied to a large area under commercial conditions. In some other experiments, seedlings were water-soaked with a heavy suspension of the causative organism. Under these conditions 1000 ppm. streptomycin sulfate was required to give approximately the same degree of control. The source of such variations might be of little concern in commercial practice, but they are of major importance in the evaluation of experimental work.

Control of bacterial spot of pepper seedlings with Agrimycin under commercial conditions

has not been attempted. However, results of parallel experiments with peppers and tomatoes indicate that the same concentration of Agrimycin is as effective on peppers as on tomatoes.

UNIVERSITY OF FLORIDA SUB-TROPICAL EXPERIMENT STATION, HOMESTEAD, FLORIDA

A RAPID METHOD OF TESTING SNAP BEANS FOR RESISTANCE TO COMMON BEAN MOSAIC VIRUS

H. Rex Thomas and H. H. Fisher

In the greenhouse it is often difficult to determine whether bean plants of certain varieties are resistant or susceptible to Common Bean Mosaic virus because of poor symptom development. This is particularly true in our breeding program when progenies involving Black Valentine, Bountiful, or Plentiful varieties are used as parents. Because of inadequate infection in the field all of our bean single plant selections are tested for resistance to Common Bean Mosaic virus in the greenhouse before planting in the field. Seed from susceptible plants is discarded. The failure to eliminate in greenhouse tests all of the susceptible selections has often resulted in seed from these plants being planted in the field and hundreds of needless selections being made. Using the strain of Common Bean Mosaic virus reported by Richards and Burkholder (3) is more satisfactory than using the common strain as it produces slightly more severe symptoms. Indexing suspected infected plants by inoculating plants of a bean variety that develops more pronounced symptoms such as Keeneys Stringless Green Refugee with the juice from the suspected plants is satisfactory, but it is time consuming and requires considerable greenhouse space.

It has been shown that under certain conditions infection with Common Bean Mosaic virus or a strain of this virus causes necrosis on resistant bean varieties whose resistance is of the dominant type derived from Corbett Refugee (2, 4, 5). Grogan and Walker (2) suggested that this response may be caused by a hypersensitive reaction of the tissue to infection. This necrotic reaction does not develop on resistant bean varieties such as Great Northern U.I. No. 1 and Robust, which have a recessive type of resistance (2, 4, 5). A factorial explanation for the resistant reaction of certain bean varieties with recessive and dominant types of resistance to infection was suggested by Ali (1).

In connection with studies of environmental factors influencing systemic necrosis in bean varieties possessing the dominant Corbett Refugee-type of resistance to Common Bean Mosaic virus it was found that a high temperature was needed for consistent systemic necrosis development. There has been developed a technique which not only gives a positive necrotic reading for virus resistance but also requires a shorter time than when it is necessary to wait for mottle symptoms.

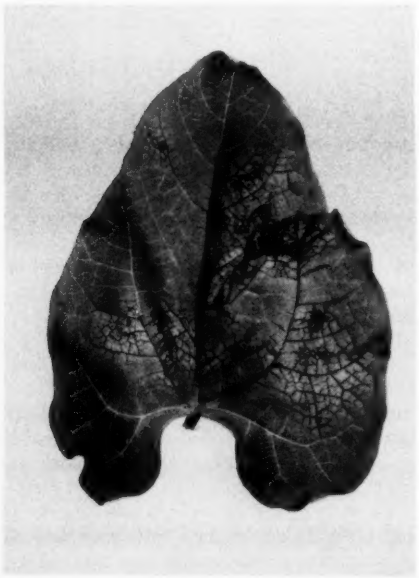


FIGURE 1. Necrosis on primary leaf of Topcrop bean (mosaic-resistant) four days after juice inoculation with Common Bean Mosaic virus. Plant was kept at 90° F.

Plants with newly expanded primary leaves are inoculated by rubbing the virus extract on leaves dusted with carborundum. After the leaves are rinsed with water, the plants are placed in a chamber maintained at 90° F. Leaf veinal necrosis usually develops on the resistant plants within three or four days (Fig. 1). No necrosis develops on the plants susceptible to the mottle type symptom. Notes can be taken at this time and all plants discarded without waiting for the development of mottling on the susceptible plants which requires seven to ten days longer.

This method of testing for resistance is not suitable when resistant plants are to be saved for seed increase or for crossing, as necrosis quickly develops in the growing point and the plant may die within a few days. Likewise this technique is not suitable for plants possessing the recessive type of resistance, such as Great Northern U.I. No. 1, as they do not develop necrosis. However, most, if not all, of the mosaic-resistant bush snap bean varieties in this country possess the dominant type of resistance derived from Corbett Refugee. This technique will also work with resistant pole beans such as Kentucky Wonder and Blue Lake, which have a dominant type of resistance probably identical with that found in Corbett Refugee.

Literature Cited

1. Ali, M. A. 1950. Genetics of resistance to the Common Bean Mosaic virus (Bean Virus 1) in the bean (Phaseolus vulgaris L.). Phytopath. 40: 69-79.
2. Grogan, R. G., and J. C. Walker, 1948. The relation of Common Mosaic to Black Root of bean. Jour. Agr. Res. 77: 315-331.
3. Richards, B. L., and W. H. Burkholder. 1943. A new mosaic disease of beans. Phytopath. 33: 1215-1216.
4. Zaumeyer, W. J., and H. Rex Thomas. 1947. Bean diseases in some of the Mountain States in 1947. Plant Dis. Reptr. 31: 432-442.
5 _____. 1948. Shiny Pod (Greasy Pod) virus and its identity with Black Root virus. (Abs.) Phytopath. 38: 29.

PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND

PRELIMINARY REPORT ON INSECT TRANSMISSION OF EASTERN PEACH X-DISEASE VIRUS IN ILLINOIS[1]

H. H. Thornberry

Studies on insect transmission of the Eastern Peach X-Disease virus were started in 1950 immediately after finding a high incidence of the disease in chokecherries in an area near Oak Forest, Illinois. Since so many of the diseased chokecherry plants showed evidence of recent infection, it seemed likely that the vector should be among the insects collected from the woody and herbaceous plants of this area. Some mass collections by sweepings were made and the insects were allowed to feed on diseased chokecherry leaves for virus acquisition before confinement to healthy foliage of peach or chokecherry seedlings for virus transmission. Particular attention was paid to leafhoppers belonging to the genus Collandonus and Scaphytopius since C. geminatus and S. acutus are vectors of the Western X-Disease Virus (2).

One of the five collections of September 14, 1950, contained four individuals of the species Collandonus clitellarius (Say)[2]. These four leafhoppers died in transit to Urbana for transmission experiments.

From the 44 mass collections of insects from vegetation in the diseased chokecherry area from June to September 1951, one collection on August 23 transmitted the disease to a peach seedling. The symptoms that developed on this seedling the following spring were typical of the disease. Identification of the disease was further confirmed by graft transmission from this diseased plant to another peach seedling. Identification[3] of the insects present in this collection revealed the following species:

Aphrophora 4-notata Say, 2.
Collandonus clitellarius Say, 1 female.
Graphocephala ? , 1 female nymph.
Jassus alitorius Say, 1 female.
Lygus oblineatus Say, 1 female.
Ormenis pruinosa, 14
Philaenus leucopthalmus L., 23

During the summer months of 1952, 36 collections were obtained from the Oak Forest area of diseased chokecherries. Peach seedlings and chokecherry seedlings exposed to the insects after a preliminary feeding on diseased chokecherry leaves have not developed symptoms of the disease. None of these collections contained any species of Collandonus. Three of the collections contained some Scaphytopius leafhoppers, probably S. frontalis[2]. A leafhopper similar to Scaphytopius (Cloanthanus) acutus (Say) has been mentioned as a suspected vector of the Eastern virus (2).

During the summer months of 1953, an intensive search was made for Collandonus leafhoppers. From some 100 collections, two Collandonus clitellarius individuals were found in sweepings from shrubs in the parks of Urbana. None was obtained in sweepings from woody and herbaceous plants in the Oak Forest area. These two C. clitellarius individuals were exposed to peach foliage showing symptoms of X-Disease. One of them confined to the diseased peach foliage in the field died within 24 hours. The other exposed to the diseased peach foliage in the laboratory for three days was transferred to healthy chokecherry seedlings. This leafhopper died within 12 hours after transfer to the chokecherry seedling. This seedling is at present dormant.

The cause of the death of the two C. clitellarius individuals confined to diseased peach tissues was not ascertainable. Death due to a toxic substance acquired from the peach tissues is suspected. Such a substance in the tissues would explain the unique situation with respect to the spread of the disease in nature, i.e. spread from chokecherry to chokecherry or peach but not from peach to peach or chokecherry (2). In such a situation the vector might survive

---

[1] Some of this information was presented to the R.A.M. Regional Technical Committee for Project NC-14 "Stone Fruit Virus Diseases and Their Control" (Minutes of the meeting at East Lansing, Michigan, March 13-14, 1953 and Annual Report for 1953).

[2] Identification by Dr. H. H. Ross, Entomolgist, Illinois Natural History Survey, Urbana, Illinois.

[3] Dr. Milton W. Sanderson, Entomolgist, Illinois Natural History Survey, Urbana, Illinois, letter of July 1, 1952.

the feeding on the toxic peach sufficiently long to transmit the virus but not long enough to acquire the virus from the peach and subsequently to migrate to a healthy plant for an inoculative feeding.

This preliminary report is published for the purpose of expediting work on the insect transmission of this disease. Although only one case of insect transmission has been achieved, the circumstances under which this single transmission was obtained and also the fact that C. geminatus is a vector of the Western X-Disease virus lead one to suspect that C. clitellarius is a vector of the Eastern X-Disease virus. This suspected vector has been identified in collections from various localities throughout Illinois (1). This distribution is illustrated in Figure 1.

FIGURE 1. The known distribution of Collandonus clitellarius (Say) by counties in Illinois according to the report by De Long (1). Counties in which the insect has been found are designated by the number of collections.

ACKNOWLEDGEMENTS

The assistance of Kenneth E. Bell, Angelos V. Adam, and Donald D. Hickman, graduate students on summer employment, in carrying out some of this work is gladly acknowledged.

Literature Cited.

1. Delong, D. M. 1948. The leafhoppers, or Cicadellidae, of Illinois. Illinois Natural History Survey Bulletin 24 (2): 97-376.
2. Stoddard, E. M., E. M. Hildebrand, D. H. Palmiter, and K. G. Parker. 1951. Virus Diseases of Peach, X-Disease (In Virus Diseases and Other Diseases with Virus-Like Symptoms of Stone Fruits in Northern America). U. S. Dept. Agr. , Agriculture Handbook 10.

UNIVERSITY OF ILLINOIS, URBANA

APPLE FRUITS A SOURCE OF OVERWINTERING FIREBLIGHT INOCULUM[1]

Robert N. Goodman[2]

In the apple producing regions of Missouri and Arkansas the predominant source of overwintering fireblight inoculum has not been definitely established. It has been difficult to attribute the occurrence of extensive fireblight infestations to the "hold-over canker" which is so rarely observed. Extensive research by Rosen (2, 3, 4) over a number of years, which included isolations from thousands of cankers and observations of thousands more, failed to demonstrate these loci of previous infections to be significant sources of primary inoculum.

On the other hand it has long been established in northern and western apple and pear producing regions that the incidence of fireblight in a given area or orchard may be directly correlated to the oozing cankers which are readily apparent in the spring.

Many fruit growers in Missouri have reported extremely severe infestations of fireblight in orchards where, seemingly, no oozing cankers had been observed. However, failure to detect these cankers does not necessarily preclude their presence.

Recently Anderson (1) demonstrated that young pear fruits could be artificially inoculated with the fireblight organism in October and serve as a source of virulent cultures of Erwinia amylovora through the following May. He suggested that infected fruit might possibly serve as a source of primary inoculum for spring infections in the absence of hold-over cankers.

On February 24, 1954, several apples of the Jonathan variety were picked from trees to which they were still adhering. All of the Jonathan trees in this block had been injured severely by fireblight during the previous spring and summer. The apples were soft, the flesh was still moist and exuded juice under slight pressure.

From tissue directly beneath the skin of these apples, virulent cultures of E. amylovora were obtained. Pathogenicity was ascertained by inoculating apple shoots on greenhouse-grown one-year-old whips of the Jonathan variety. Isolations from shoots thus infected also provided virulent cultures.

Literature Cited

1. Anderson, H. W. 1952. Maintaining virulent cultures of Erwinia amylovora and suggestion of overwinter survival in mummied fruit. Plant Dis. Reptr. 36: 301-302.
2. Rosen, H. R. 1929. The life history of the fireblight pathogen Bacillus amylovorus, as related to the means of overwintering and dissemination. Arkansas Agr. Expt. Sta. Bull. 244.
3. _____. 1929. The relationship of different species of pomaceous hosts to the overwintering of the fireblight germ. Arkansas Agr. Expt. Sta. Bull. 246.
4. _____. 1936. Oversummering of fireblight pathogen, spraying for control of fire blight, and abscission induced by Erwinia amylovora and Phytomonas syringae. Arkansas Agr. Expt. Sta. Bull. 330.

UNIVERSITY OF MISSOURI, COLUMBIA, MISSOURI

[1] Journal Series Paper No. 1428 approved by the Director of the Missouri Agricultural Experiment Station.

[2] Assistant Professor of Horticulture, Department of Horticulture, University of Missouri.

CHALAROPSIS THIELAVIOIDES PEYRONEL FOUND ON EVERGREEN GRAFTING STOCK

Charles E. Hess and D. S. Welch

Chalaropsis thielavioides Peyronel, the pathogen which causes "black mold" on rose grafts, has been identified as the cause of graft failure in several evergreen plants. The fungus was first described and named in 1915 by Peyronel in Italy where it caused a root rot of lupine (6). Subsequent reports include failure of walnut grafts in England in 1930 (3), presence of the fungus in Palestine on peach seedlings imported from France in 1933 (3), and isolation from rose cuttings in Australia in 1936 (5). The first isolation of Chalaropsis thielavioides in North America was from root rot on Chinese elm in Oklahoma in 1934. A serious outbreak occurred in the Eastern States and in the vicinity of Chicago on rose grafts in 1938 and 1939 (4). In 1945 black mold caused failure of bud unions in several rose fields and of grafts in a greenhouse in California (2). It reappeared in California in 1953 causing a root rot of greenhouse roses (1).

The present report deals with specimens sent from a nursery in New Jersey. Both grafted plants and cuttings were affected. In all cases callus formation had been prevented and the cut surfaces were covered with the characteristic black, crumbly masses of macroconidia.

A loss of 100 percent was reported for grafts and cuttings of Cryptomeria japonica D. Don. (Cupressus japonica L.). Severe losses occurred in grafts of Ilex opaca Ait., Thuja orientalis L. (Biota orientalis Endl.), and Thuja occidentalis L. A moderate loss was sustained in Juniperus virginiana L. grafts, although losses the previous year were severe in this species.

Black mold was first noticed in the greenhouses early in 1953, occurring on Ilex opaca, Juniperus virginiana, and Thuja occidentalis. During the summer of 1953 the greenhouses were cleared of all plant materials and the walls, benches, and floors were thoroughly sprayed with formaldehyde in an attempt to eradicate the pathogen. In the fall the understock was brought into the greenhouses, trimmed and potted, and placed in the grafting benches to reroot before the grafting operation. It was noticed that the wounds made in trimming the Cryptomeria japonica did not callus over and in a few cases black spore masses were seen. However, the understock was used and approximately four weeks after grafting the cut surfaces at the unions on several plants were covered with black spores. The same condition developed on more and more plants until the entire lot of grafts was affected. The plants were removed and burned. Ilex opaca, adjacent to the Cryptomeria, also became affected and later black mold was found on Juniperus virginiana and Thuja orientalis and T. occidentalis in other parts of the greenhouse. Since the wounds of the Ilex, Juniperus, and Thuja callused normally, and in view of the thorough formaldehyde treatment which the house had received, the probable source of inoculum was the Cryptomeria understock.

The symptoms and signs of Chalaropsis thielavioides on evergreens are very similar to those described for black mold on rose grafts. There is little or no formation of callus. In the early stages of the disease there is an inconspicuous formation of whitish, mildew-like mycelium and conidiophores bearing endoconidia on the cut surfaces of the union. This white growth gradually darkens and becomes more conspicuous with the formation of the macroconidia which give the characteristic black mold appearance.

SUMMARY

1. Chalaropsis thielavioides has been identified as causing graft failure of Cryptomeria japonica, Ilex opaca, Juniperus virginiana, Thuja orientalis, and Thuja occidentalis in a New Jersey nursery.

2. The probable source of inoculum was understock of Cryptomeria japonica.

Literature Cited

1. Baker, K. F. 1953. Chalaropsis root rot of rose. Plant Dis. Reptr. 37: 430-433. 37: 430-433.
2. _____, and H. E. Thomas. 1946. Failure of bud and graft unions of rose induced by Chalaropsis thielavioides. Phytopath. 36: 281-291.
3. Hamond, Joyce B. 1935. The morphology, physiology and mode of parasitism of a species of Chalaropsis infecting nursery walnut trees. Jour. Pomol. 13: 81-107.
4. Longree, K. 1940. Chalaropsis thielavioides, cause of "black mold" of rose

grafts. Phytopath. 30: 793-807.

5. Mason, E. W. 1941. Annotated account of fungi received at the Imperial Mycological Institute. List II, Fascicle 3 -- Special Part: 131-133. Commonwealth Mycol. Inst., Kew.
6. Peyronel, F. 1916. Una nuova malattia del lupino prodotta da Chalaropsis thielavioides Peyr. nov. gen. et nova sp. Staz Sper. Agr. Ital. 49: 583-596.

NEW YORK AGRICULTURAL EXPERIMENT STATION, ITHACA

DISSEMINATION OF PLANT PATHOGENS ON NURSERY AND ORNAMENTAL PLANTS

Roy A. Young

Few agriculturists face as many plant disease problems as confront nurserymen, for in no other agricultural industry are so many different species and varieties of plants grown so intensively on small acreages. In spite of the nurseryman's familarity with plant diseases, the sudden appearance of a new disease on a specialty crop that has been grown for years in an area always causes concern. Questions continually arise as to the origin of new diseases and as to why there are so many more diseases now than a few years ago. Undoubtedly increased knowledge, leading to improved recognition, has contributed largely to the apparent increase in the number of diseases. Many effects that were previously attributed to drought, sunburn, or poor cultural conditions are now known to be caused by specific plant pathogens. It is likely also that a few new pathogens may have developed through genetic recombination or mutation. By far the greater proportion of the apparently new diseases, however, may be attributed to wider distribution of old diseases due to increased shipment of plants and plant materials and to intensification of agriculture leading to closer proximity of plantings and more frequent land use.

In the nursery industry, plants and plant materials are shipped all over the world. Instead of seed, whole plants or vegetative parts suitable for propagation are shipped, thus the opportunities for disseminating agents of infection are greatly increased. The following examples, based on observations in Oregon, are cited to illustrate the ease with which agents of plant disease may be disseminated in association with nurserystock, and the serious consequences of such dissemination.

CAMELLIA FLOWER BLIGHT: Prior to 1940 there were no virulent fungus or bacterial pathogens of camellia foliage or flowers present in Oregon. Now the camellia flower blight pathogen (Sclerotinia camelliae Hara) is established in several locations in the Willamette Valley. The disease was first reported in Japan in 1919 and was probably brought to California with imported nurserystock (5, 10). Flower blight was observed in a nursery in California in 1938 and apparently spread from there to other Pacific Coast nurseries and to a number of nurseries in the Southern States. The pathogen is an ascomycetous fungus that survives unfavorable periods in the form of a sclerotium that develops in the base of infected flowers. These sclerotia may remain viable in the soil for several years. Each year when camellias are in bloom apothecia develop from the sclerotia and discharge ascospores that may infect camellia flowers. A single sclerotium has a tremendous infection potential. Many apothecia, each containing thousands of spores, may develop from a sclerotium over a period of years. Thus a single imported, balled or potted camellia plant with sclerotia in the soil around its roots might serve for several years as a source of infection for flowers on nearby plants.

Preventing spread of this pathogen by regulatory measures is very difficult. When inspecting canned or balled camellia plants for the disease, it is possible to detect old diseased flowers on the surface of the soil very readily; however, it is almost impossible to find small portions of sclerotia in the soil without disturbing the plants too greatly. In view of the difficulty of detecting sclerotia of the pathogen in the soil an effort has been made to avoid introducing the pathogen into States where it is not present by restricting shipment of plants with soil around the roots. While small plants can be shipped satisfactorily with bare roots, larger plants cannot tolerate the drying involved.

Elimination of this pathogen from an area is extremely difficult due to the longevity of the sclerotia and the infection potential of a single spore cup. Results from attempts at chemical control have not been satisfactory. Extreme sanitation involving the removal of all dead flowers to prevent entrance of sclerotia into the soil is the most promising control (13).

CYPRESS ROOT ROT: During the past three years the root rot disease of Lawson cypress (Chamaecyparis lawsoniana Parl.), caused by Phytophthora cinnamomi Rands, has been investigated extensively in Oregon. The pathogen, an introduced organism, has become established in the Southeast and some other parts of the United States and was probably brought into Oregon on infected nursery stock. On Lawson cypress the pathogen infects secondary roots, and progresses into the lower stem of the host, killing root tissues as it advances. Commonly foliar symptoms do not develop until the pathogen has progressed into the crown of an infected plant. Infected plants eventually die as a result of loss of roots and girdling of the stem. The pathogen has been transferred to nurseries and landscape plantings either in

infected roots or infested soil and at present is known to occur to a limited extent in at least nine counties in Oregon (11).

The importation of P. cinnamomi serves well to illustrate the seriousness of the danger of unknowingly disseminating pathogens when shipping nursery stock. The presence of the fungus in in-transit nursery stock cannot be detected by practical plant quarantine inspections since the fungus may be carried in the roots of apparently healthy plants or in the soil surrounding the roots of healthy plants. Frequently, foliar symptoms are not apparent until the root system of an affected plant is almost destroyed. By removing the soil from around the roots of balled plants infected roots can usually be detected since healthy roots are bright red as contrasted to the dull brown of diseased roots. Such thorough inspections are not practical, however, and even if such a check were made, incipient infections could not be detected nor could the presence of the fungus in the soil around roots of healthy plants be determined.

Once established in a nursery P. cinnamomi is extremely difficult, if not impossible, to eradicate. The fungus may survive in the soil for long periods of time in the absence of a susceptible host; therefore, rotation of crops is of little use as a measure to reduce the incidence of the pathogen in the soil. In Oregon nurseries the pathogen has been recovered from depths down to 30 inches, well below the effective range of most chemicals used as soil fumigants. Since the fungus does not die out of infested soil readily and cannot be eliminated chemically, it has been necessary for growers of Lawson cypress to move to new land.

The fact that P. cinnamomi attacks a wide range of host plants increases the difficulty of preventing dissemination of the pathogen. White birch, bruckenthalia, heather (eight species of Erica and two varieties of Calluna vulgaris), chestnut, Lawson cypress, walnut, Douglas fir, mollis azalea, rhododendron, Irish and Japanese yew, were among the plants experimentally proven to be susceptible to infection by P. cinnamomi (11).

Although the pathogen is most severe on cypress, yew, and heather, it infects the small feeder roots of all of the plants listed above and will eventually kill young plants of most of these species. Older plants frequently are not killed but may be severely injured as a result of the destruction of secondary roots. Economically this pathogen causes large losses even in varieties of plants that are not killed, since most nursery stock is priced on the basis of size and any retardation of growth represents an economic loss. The wide host range facilitates the spread of the pathogen locally to other nurseries and to home landscape plantings.

GLADIOLUS DISEASES: The methods of culture and handling involved in commercial production of gladiolus corms and flowers greatly facilitate the rapid dissemination of all types of diseases of gladiolus. Gladioli are propagated from vegetative structures, corms, that serve well as a means of perpetuating and disseminating fungus, bacterial, and virus diseases. Gladiolus corms are shipped all over the world. Small corms may be imported from Holland, increased in Oregon for one year, and shipped to Florida for production of cut flowers. Corms from New York may be shipped to Oregon to be grown on consignment for eventual production of flowers in the Southeastern United States.

Of the diseases spread in association with gladiolus corms, probably the virus diseases constitute the most striking example. Control of virus diseases in many of our annual plants is greatly aided by the failure of many viruses to survive in the seed and be transmitted to a new generation. However, seeds are not used in gladiolus propagation other than in production of new varieties, since most commercial gladioli are hybrid in origin. Therefore vegetative reproductive parts are used in propagation in order to maintain trueness to variety. When a gladiolus plant becomes infected with a certain virus, the cormels from that plant may be infected and may serve as sources of inoculum from which the virus may spread to other plants. Since corms are shipped to many parts of the world a certain stock of infected corms may serve as a reservoir of infection from which plantings of gladioli in many areas of the world may become infected.

Those virus diseases that cause striking symptoms on gladiolus foliage and flowers may be detected in plantings and an effort made to eliminate infected corms. However, there are viruses that may be present in gladiolus where they cause little or no apparent effect but may be devastating in their effect on other crops grown in close proximity.

Bean Yellow Mosaic (Bean Virus 2) may have little or no effect on gladioli, but when gladioli are grown in fields adjacent to pole beans most of the bean plants within a radius of 75 to 100 feet may be severely dwarfed and mottled (8, 9). Other viruses known to be carried in gladioli are strains of the Tobacco Ring-spot (3, 4), Tobacco Mosaic (3), Cucumber Mosaic (4), and Aster Yellows (7, 9) viruses. Of these viruses only Aster Yellows appears to be lethal to gladiolus, while all may cause serious damage to other host plants. Preventing increase and

dissemination of these viruses is very difficult since, with the possible exception of the Aster Yellows virus (7), there are no known symptoms by which infected corms can be distinguished from healthy corms.

There are also a number of fungus-induced gladiolus corm diseases that may be spread in association with infected corms. The most common of these diseases are caused by Fusarium species and by Stromatinia gladioli (Drayton) Whet. (Sclerotinia gladioli Drayton). These pathogens may cause obvious symptoms on infected corms or may be carried in association with gladiolus husks and not be detectable by macroscopic observation. These fungi are classified as soil inhabitants since they can survive in soil for long periods in the absence of the gladiolus host. Once an area of soil becomes infested, corms planted in the soil for several years afterward may become infected. The latest gladiolus pathogen of this type to be introduced into Oregon was Curvularia lunata (Wakker) Boedijn, which was first reported in December 1952 (2). This pathogen has been prevalent in Southeastern States for several years and was probably introduced on corms shipped from that area.

CROWN GALL: The development of crown gall, caused by Agrobacterium tumefaciens (E. F. Sm. & Townsend) Conn, annually causes losses to nurserymen who propagate tree fruit nursery stock. Losses of fruit and flowering cherry stock ranging up to $1200 per acre were reported in 1949, 1950, and 1951 by Oregon nurserymen. Although this disease frequently is not seriously debilitating, the more severe infections may result in complete girdling of the main root or crown of a young tree by gall tissue, and marked retardation of growth. In spite of the absence of harmful effects from smaller galls, particularly on lateral roots, the presence of a gall subjects the plant to quarantine and plants with crown gall are therefore unsalable.

It was observed in Oregon that Mazzard Cherry seedlings from one source might develop a high incidence of crown gall when lined out as root stocks for budding or grafting while seedlings from another source might be relatively free from gall when grown similarly in adjacent blocks in the same soil (12). Thus, it appeared that young seedlings were infected at the time that they were lined out. Greenhouse testing of cherry seedlings grown for use as understock showed that frequently many seedlings are infected when they are planted in nurseries for budding or grafting. As many as 26.8 percent of one lot of seedlings developed crown gall when grown in sterile soil, and in four other lots 11.0, 10.6, 8.2, and 6.9 percent of the seedlings were similarly found to be infected. These seedlings apparently had crown gall bacteria within their roots when dug or had incipient galls initiated but not detectable. Based on 1952 prices a 10 percent loss in sale price resulting from use of infected seedlings would amount to from $700 to $1800 per acre depending on the nature of the nurserystock. These loss figures would not include expenses involved in the two or three years of culture necessary to produce the salable product.

ROOT LESION (MEADOW) NEMATODES: Since the root lesion nematodes (Pratylenchus spp.), are root parasties they are not disseminated on seed. However, they may be carried from one area to another in the soil around balled nursery stock and in the roots of nursery stock. These nematodes may seriously damage many types of nursery plants and cause large losses to nurserymen. In an Oregon nursery a severe infestation on boxwood was observed that was directly traceable to an out-of-state nursery where the boxwood had originally been grown. The meadow nematodes in the Oregon nursery were confined to an area of soil where approximately 6,000 boxwood plants from another State had been planted. Similarly, root lesion nematodes were found in the roots of most of the plants examined in two shipments of Christmas rose (Helleborus niger L.) from Holland (1).

Severe infestations of root lesion nematodes have also been observed on Mazzard cherry seedlings (14) and daffodils (6). The nematodes affect the small feeder roots of the Mazzard cherry seedlings and greatly retard growth of the young plants. The species found associated with the roots of cherry seedlings were also found in the roots of numerous weed and cover crop plants grown after the cherries had been removed. Alsike clover, crimson clover, oats, black bindweed, Brewer's bitter cress, common chickweed, dog fennel, lambs-quarter, and wild radish were moderately to heavily infested with root lesion nematodes (14).

The wide host range would indicate that these parasitic nematodes would find ample nutrition to sustain them until another crop of cherry seedlings was grown in the same area. In a situation of this type it would probably be necessary to fumigate the soil with a volatile chemical in order to eliminate the nematodes from the area.

CONCLUSION: These examples of dissemination of plant pathogens in association with

nursery stock are cited to illustrate the importance of the effect of such dissemination on the development of serious nursery diseases. It is obvious that shipping point and terminal inspections alone will not suffice to prevent the spread of plant pathogens in association with nursery stock, for in many cases it is not possible to determine by such inspections whether or not pathogens are present. As agriculture becomes more and more intensive it will probably become necessary to increase the attention given to field inspection of nursery crops and also to develop efficient methods of disease indexing so that disease-free propagating stock can be grown. This is particularly true of such crops as gladiolus, in which certain viruses may be present in corms without recognizable symptoms.

Literature Cited

1. Anderson, C. G., and H. J. Jensen. 1952. Root-lesion nematodes present in Christmas rose imported from Holland. Plant Dis. Reptr. 36: 253.
2. _____, Roy A. Young, and E. T. Palm. 1952. Curvularia on gladiolus in Oregon. Plant Dis. Reptr. 36: 474.
3. Berkeley, G. H. 1951. Gladiolus viruses. (Abstr.) Phytopath. 41: 3-4.
4. Bridgmon, G. H. 1951. Gladiolus as a virus reservoir. (Abstr.) Phytopath. 41: 5.
5. Hansen, H. N., and H. Earl Thomas, 1940. Flower blight of camellias. Phytopath. 30: 166-170.
6. Jensen, Harold J., C. G. Anderson, and J. Wieman. 1951. A root-lesion nematode disease of narcissus. Plant Dis. Reptr. 35: 522-523.
7. Magie, R. O., Floyd F. Smith, and Philip Brierley. 1952. Occurrence of western aster yellows virus infection in gladiolus in eastern United States. Plant Dis. Reptr. 36: 468-470.
8. McWhorter, F. P., L. Boyle, and B. F. Dana. 1947. Production of yellow bean mosaic in beans by virus from mottled gladiolus. Science 105: 177-178.
9. Palm, E. T., and Roy A. Young. 1952. Observations of gladiolus abnormalities in Oregon in 1951. Plant Dis. Reptr. 36: 108-109.
10. Thomas, H. Earl, and H. N. Hansen. Camellia flower blight. Phytopath. 36: 380-381.
11. Torgeson, D. C., Roy A. Young and J. A. Milbrath. Phytophthora root rot diseases of Lawson cypress and other ornamentals. Oregon Agr. Exp. Sta. Bull. 537. January 1954.
12. Young, Roy A. 1952. Incipient crown gall infections in Mazzard cherry seedlings. (Abstr.) Phytopath. 42: 520.
13. _____, and J. A. Milbrath . 1950. Camellia flower blight. American Camellia Yearbook. 254-261.
14. _____, D. C. Torgeson, and C. G. Anderson. 1950. Meadow nematodes (Pratylenchus sp.) on Mazzard cherry and forage plants and weeds in nursery rotations. Plant Dis. Reptr. 34: 230-231.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON STATE COLLEGE, CORVALLIS, OREGON

UNUSUAL RECORDS OF PLANT DISEASES

OUTBREAK OF PYTHIUM ROT IN NEWLY SET FLUECURED TOBACCO IN VIRGINIA

By Wilbert A. Jenkins

Soft, watery rots affecting the newly set fluecured tobacco crop in Virginia have reached essentially epiphytotic status during the current transplanting season. Losses in stands have varied from minor to proportions that have necessitated plowing up and resetting several acres of land. Such losses, particularly in conjunction with the grower's apprehension of black shank, have developed widespread interest and uneasiness.

Contrary to our findings in 1949, Pythium rot during the current season is causing rather typical "damping-off" symptoms, rather than bud rotting. The rot is mostly confined to the upper hypocotyl and lower stem regions, bringing about complete collapse of the plant in this area. The lesion varies in color from intense black to brown, and rarely extends into the lower hypocotyl or into the stem above the soil level. The exterior of the lesion is commonly slimy to the touch and extremely watery. Occasionally there is some retting of the vascular tissues.

Microscopic examination of affected tissues reveals rather typical Pythium mycelium in most cases. In some instances the appearance of the mycelium is more like a Phytophthora except for general diameter of hyphae. Prevailing low temperatures have not favored sporulation, thus making specific determinations difficult. However, it seems certain that several species of Pythium are concerned, among them being P. debaryanum, P. aphanidermatum, and perhaps P. arrhenomanes. These tentative determinations are being checked by more complete culture procedures.

The severity of the disease this season appears to be due basically to the unusually wet cold weather which has persisted since about May 3. Additional contributing factors are mechanical injuries sustained by the seedlings either before or following transplanting. Seedlings in this area were unusually tender though of transplanting size, and were rather seriously bruised and crushed during the pulling operation. There has been little flea beetle injury as compared with last season, but wireworm damage has been extensive. In addition, the factors conducive to slow rooting of the transplants have apparently favored excessive water congestion in the tender stems, which is not relieved by normal transpiration and thus results in partial suffocation of tissues. These factors, and probably others, have undoubtedly contributed heavily to rapid invasion by species of Pythium, which were afterwards afforded almost ideal temperature and moisture relationships for further development.

TOBACCO RESEARCH STATION, CHATHAM, VIRGINIA

TOBACCO PLANT SITUATION IN SOUTHWEST VIRGINIA

By S. B. Fenne

On April 27, 28, and 29, tobacco plant beds were visited in Lee, Wise, Dickinson, Buchanan, Russell, and Washington Counties in the burley area of southwest Virginia. No plant diseases were found and many very good plant beds were seen. In some beds, however, there was definite injury to plants in portions of the beds. Usually this injury occurred in low spots. Plants in affected areas were much smaller. The leaves were often yellow and in many cases they were cupped and considerably thicker than normal. Nematodes frequently were found on the roots of damaged plants examined in the laboratory.

It appeared that the injury observed in these plant beds was probably due to the excessive accumulation of plant nutrients. Many of these beds had been treated with cyanamid, or fertilizers had been used at excessive rates. In many cases plants were stunted or retarded. A common practice in Southwest Virginia is to use a regular field fertilizer in plant beds. Such fertilizers contain a considerable amount of chlorine.

VIRGINIA POLYTECHNIC INSTITUTE EXTENSION SERVICE, BLACKSBURG

POTATO WART INTERCEPTED FROM BOLIVIA

By Paul X. Peltier

A shipment of potato tubers for propagation arriving in the mail from Bolivia at the Washington, D. C, Inspection House en route to Orono, Maine on April 4, 1954, was found infected with potato wart, Synchytrium endobioticum (Schib.) Perc., by W. B. Wood. One infected potato was found in a shipment of 13 assorted tubers of various sizes, shapes and

colors that seemed to consist of at least three varieties. The entire shipment of potatoes was destroyed.

This is our third interception of potato wart. The two previous interceptions were made at the D. C. Inspection House in 1932 on potato tubers from Bolivia and Peru by D. P. Limber.

U. S. DEPARTMENT OF AGRICULTURE, AGRICULTURAL RESEARCH SERVICE, PLANT QUARANTINE BRANCH, WASHINGTON, D.C.

DESTRUCTIVE OUTBREAK OF LOOSE SMUT IN A GEORGIA WHEAT FIELD

By T. D. Persons

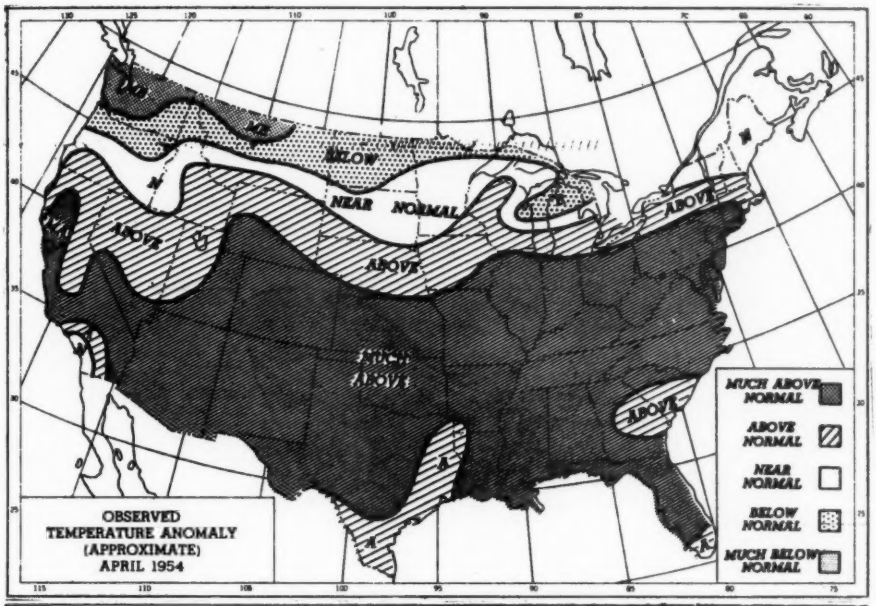
Mr. Thomas Duck, Jr., of the White Fringed Beetle Control Project recently brought in some specimens of wheat from a farm in Jones County, Georgia infected with loose smut (Ustilago tritici). Mr. Duck informed me that this field, about 40 acres, is so severely affected that there will be a total loss of grain.

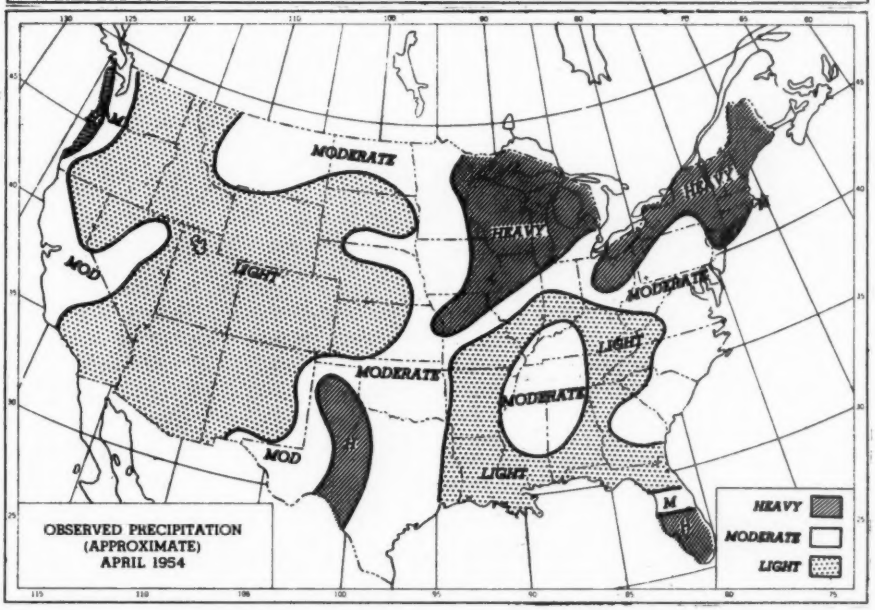
I have observed varying degrees of loose smut infections in a number of other fields of wheat in the central Georgia area this season, but none of them even approached this one in severity.

PHONY PEACH AND PEACH MOSAIC CONTROL, U. S. DEPARTMENT OF AGRICULTURE, AGRICULTURAL RESEARCH SERVICE

PROCEEDINGS OF THE SEVENTH INTERNATIONAL BOTANICAL CONGRESS

After a long delay due to circumstances beyond the control of the editors, the Proceedings of the Seventh International Botanical Congress, held in Stockholm, Sweden, in July 1950, have been published. The single volume of 899 pages contains an adequate account of the organization of the Congress, the several plenary sessions, the many botanical excursions, and gives a complete list of delegates. Abstracts of papers read before the 14 sessions other than nomenclature are included and the general papers presented at the plenary sessions are published in full. A very complete account of the proceedings of the nomenclature session is of particular interest. The volume is published by Almqvist and Wiksell, Stockholm, Sweden. The Chronica Botanica Co., Waltham, Massachusetts, is the American agent.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.